THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—13TH YEAR. SYDNEY: SATURDAY, SEPTEMBER 11, 1926. No. 11.

Table of Contents

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

ORIGINAL ARTICLES— PAGE.

"Albuminuria of Pregnancy, Preeclamptic Toxæmia and Eclampsia, with Special Reference to the Estimation of the Urea Content of the Blood," by A. J. CUNNINGHAM, M.D., Ch.M. .. 331

REPORTS OF CASES—

"Cyst of the Astragalus," by R. B. WADE, M.D., Ch.M., AND KEITH INGLIS, M.D., Ch.M. .. 345

"Hæmangio-Lymphangioma of the Abdominal Wall and External Genitals in a Child," by ARCHIE ASPINALL, M.B., Ch.M., AND KEITH INGLIS, M.D., Ch.M. .. 345

"Aneurysm of the Hepatic Artery Associated with Acute Endocarditis," by E. H. STOKES, M.B., Ch.M., AND KEITH INGLIS, M.D., Ch.M. .. 346

REVIEWS—

Andreas Vesalius .. 351
Emergency Surgery .. 352
A Textbook on Physiology .. 352
Oto-Rhino-Laryngology for Nurses .. 352

LEADING ARTICLES—

Passive Acquiescence .. 353

CURRENT COMMENT— PAGE.

Eclampsia .. 354
The Use of "Lipiodol" in the Spinal Theca .. 355

ABSTRACTS FROM CURRENT MEDICAL LITERATURE—

Dermatology .. 356
Radiology .. 357

BRITISH MEDICAL ASSOCIATION NEWS—

Scientific .. 358

THE AUSTRALASIAN MEDICAL PUBLISHING COMPANY, LIMITED—

Annual Meeting .. 363

OBITUARY—

Charles Vance Smith .. 364
Sidney Vere Appleyard .. 364
Frederick John Thomas Sawkins .. 364
John Theodore Anderson .. 364

MEDICAL APPOINTMENTS .. 364

MEDICAL APPOINTMENTS VACANT, ETC. .. 364

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 364

DIARY FOR THE MONTH .. 364

EDITORIAL NOTICES .. 364

ALBUMINURIA OF PREGNANCY, PREECLAMPTIC TOXÆMIA AND ECLAMPSIA, WITH SPECIAL REFERENCE TO THE ESTIMATION OF THE UREA CONTENT OF THE BLOOD.[1]

By A. J. CUNNINGHAM, M.D., Ch.M. (Sydney),
Sydney.

INTRODUCTION.

ALBUMINURIA of pregnancy, preeclamptic toxæmia and eclampsia are sometimes described as three separate conditions. They are also grouped together as associated diseases. Furthermore they are occasionally considered to be different stages of one disease. There seems to me to be sufficient evidence that the latter is the correct view. If such be the case, albuminuria with headache or with œdema would be the earliest manifestation and persistent convulsions and coma would be the terminal stage.

Albuminuria is the only constant manifestation of this disease. When I say constant I realize that sometimes an examination of the urine during eclamptic conditions does not reveal albumin, although albumin will appear eventually in every case after repeated examinations.

Throughout this paper these three conditions will be referred to as far as possible as albuminuria of pregnancy.

An attempt will be made to demonstrate the value of blood urea estimations: (i.) As regards diagnosis, especially differentiating albuminuria due to a toxæmia from that due to chronic interstitial nephritis; (ii.) as regards prognosis. Its greatest value lies in this direction.

In discussing this toxæmia of pregnancy (albuminuria, preeclamptic state and eclampsia) we are inevitably drawn to a consideration of two other groups of cases which are very closely allied to this condition. These are chronic interstitial nephritis (during which toxæmic symptoms and even eclamptic convulsions are very prone to occur) and uræmia.

Thus we have three large groups whose chief common manifestation is the presence of albumin in the urine: (i.) Albuminuria of pregnancy; (ii.) nephritic toxæmia; (iii.) uræmia.

[1] Accepted as a thesis for the degree of doctor of medicine at the University of Sydney.

The groups merge into one another and it is often difficult to define a case accurately as belonging to one group. Any observation (clinical or chemical) that is going to help in this direction, is of great value, especially from the point of view of prognosis and treatment.

I not only propose to discuss the first group but also the second group.

DEFINITION.

Albuminuria of pregnancy may be defined as a condition of pregnancy due to a toxæmia, sometimes terminating in convulsions (which may occur before, during or after labour) and whose outstanding manifestation is albuminuria.

ÆTIOLOGY.

The cause of albuminuria of pregnancy is unknown. There have been many theories formulated and an enormous amount of research has been carried out in an attempt to determine the causal factor.

The only positive fact is a characteristic pathological lesion which has been found in women who have died following eclamptic convulsions. This consists in characteristic hæmorrhages into the liver and other important organs and does not occur in any other conditions. The most one can say is that the disease is a profound toxæmia whose origin is unknown.

The nature of the poison is also unknown, but in its action it resembles certain organic poisons, particularly the poisonous albumins, proteoses, albuminoses and peptones (snake venom), certain bacterial poisons and in some respects chloroform poisoning.

THEORIES OF CAUSATION.

A prevalent view (first put forward by Veit) is that there is normally an intoxication by trophoblastic elements (a syncitiotoxin produced by syncitiolysis) which is neutralized by antibodies produced by the maternal organism. The failure of this reaction results in a toxæmia. This is supported by the following facts:

(i.) The toxæmia never occurs apart from pregnancy.

(ii.) It is relieved by labour at term or at the end of pregnancy at any time.

(iii.) The pathological findings are characteristic and are not found apart from pregnancy.

There are three possible sources of origin: placental, maternal and fœtal.

The Placental Theory.

(*a*) It is known that portions of the trophoblast may be found in the maternal circulation in the liver and in other organs.

(*b*) Abderhalden claims to have shown there is present in the blood of pregnant women a ferment capable of digesting placental albuminoses.

(*c*) The associations with hydatidiform mole and with recent death of the fœtus strongly favour this theory.

(*d*) It has been stated by Schmorl and others that proliferation of the chorionic epithelium with the formation of numerous trophoblastic emboli is met with in cases that terminated in eclamptic convulsions.

(*e*) Some observers cite the fact that injection of placental extract derived from women who have had eclamptic convulsions or even from normal gravid women has produced similar convulsions in rabbits, but the injection of other material has produced the same results.

(*f*) The fact that there is recent infarction of the placenta strongly supports this theory. Young considers that the poison is liberated into the maternal circulation by infarction of the placenta. Whether this be true or not, the fact that there is recent infarction of the placenta suggests that the origin of the poison may be at the placental site.

Acting from instructions from the Professor of Obstetrics I have washed out many placentæ from this type of case. All showed infarcts, either old or recent. One rather interesting specimen was from a woman who had definite preeclamptic symptoms just prior to delivery. This placenta contained two or three old infarcts and one large recent infarct. The recent infarct apparently corresponded to the preeclamptic symptoms.

Against this theory are brought the cases of *post partum* eclampsia and also those rare cases associated with ruptured ectopic gestation. The fact that convulsions always occur within forty-eight hours and generally within six hours of delivery and that they nearly always occur in patients who have had no antenatal treatment, suggest that the first argument against this theory can be refuted. It is also conceivable that the length of time from the moment when the poison enters the maternal circulation till convulsions occur varies in different subjects.

Again those cases in which convulsions begin during labour and still continue after delivery, are cited against this theory. These are generally believed to be serious. I have seen several of this type of case and it seems to me that the nature of the convulsions and the condition of the patient differ somewhat from the true eclamptic stage. The patient is more irritable and the preliminary twitching is much diminished or entirely absent. I believe that the convulsions that continue after delivery, are due to small cerebral hæmorrhages and the patient recovers or dies according to the extent of the hæmorrhages. Other theories will not be discussed fully.

Theories of Maternal Origin.

(*a*) The maternal excretory organs are overtaxed. The patient is poisoned by her own and the fœtus's waste products. This theory presupposes some renal insufficiency.

(*b*) According to the mechanical theory the ureters are dilated, especially the left one, during the last trimester of pregnancy.

(*c*) According to Selheim's theory the toxæmia is due to mammary activity, the condition being apparently parallel to the milk fever of cattle.

(*d*) According to the Zwiefel theory the toxæmia is due to the presence of sarco-lactic acid which he has found in the blood and cerebro-spinal fluid of women with eclamptic convulsions.

(*e*) Thyreoid and parathyreoid insufficiency have been suspected of being the cause.

Theories of Fœtal Origin.

(*a*) The fœtus produces the causal toxin. This is negatived by the fact that eclampsia occurs in hydatidiform mole and also the advent of toxæmia is frequently synchronous with the death of the fœtus.

(*b*) The mother is sensitized during pregnancy by small quantities of fœtal protein and would develope anaphylactic shock if a quantity of fœtal blood were suddenly introduced into the circulation.

Infection at the placental site has been put forward as a cause. There is nothing to support this.

Albuminuria of pregnancy is more common in *primiparæ* and especially in very young *primiparæ*. Twins and hydramnios seem to predispose to it. It is sometimes associated with hydatidiform mole. Toxæmic symptoms commonly supervene on chronic renal disease. Albuminuria of pregnancy generally occurs after the seventh month, but eclamptic convulsions have been noted as early as the tenth week.

The convulsions may be *ante partum*, *intra partum* and *post partum*. The percentage incidence has been stated as 30%, 50% and 20% respectively. Probably a greater percentage is *ante partum* than is stated above. It is well known that the onset of labour or premature labour very often closely follows the onset of convulsions. Thus the majority of patients with *intra partum* eclampsia admitted to large hospitals were probably really suffering from *ante partum* eclampsia.

A definite type of patient is recognizable. She is fair haired, stout and with heavy features and limbs.

Post partum convulsions usually arise within six hours after delivery. When convulsions have ceased for a day or two they rarely recur.

Recurrence of albuminuria in subsequent pregnancies is not uncommon, but the recurrence of eclamptic seizures is rare.

Sudden changes in climate in the form of a rapid fall of temperature, injudicious diet and constipation may precipitate an attack.

The toxæmia is not due to renal changes, but if such be present, they play an important part in aggravating the condition. This supports the view that the condition is due to a poison which is excreted by the kidneys, bowel and skin.

Heredity has been stated to be a predisposing factor.

Pathology.

The pathological findings of the toxæmia are characteristic and do not occur in any other known condition.

The liver is swollen and pale. There are areas of subcapsular hæmorrhage which are occasionally extensive. This has been suggested as an explanation of the severe epigastric pain which occurs in the preeclamptic stage in about 60% of cases.

There are hæmorrhages in the liver substance. Microscopically there is universal granular and fatty degeneration of the protoplasm of the liver cells. Focal areas of hæmorrhage and necrosis are present. The areas of necrosis involve the periphery of the individual lobules and the portal spaces and are probably due to thrombotic processes in the smaller portal vessels.

In the kidneys there is parenchymatous degeneration of varying degrees of severity. The kidney is large and the cortex swollen and finely granular on section. The blood vessels are distended. Focal necrosis with hæmorrhage may be found. Microscopically the highly specialized cells of the convoluted tubules and the ascending loop of Henle bear the brunt of the attack, all stages of cell degeneration being seen. These changes are probably purely secondary.

Dilatation of the upper part of the ureters and of the renal pelvis were stated to be a cause of albuminuria of pregnancy, but dilatation of the right ureter above the brim of the pelvis and of the renal pelvis is a normal pregnant condition. Dilatation of the left ureter and corresponding dilatation of the left renal pelvis is present to a less extent in a high proportion of pregnant women.

The muscle of the heart is soft, pale and moist; subendocardial hæmorrhages may be found. Microscopically there is universal granular degeneration and occasional fatty degeneration of the protoplasm of muscle cells.

The brain manifests flattening and œdema of the convolutions, sometimes with hyperæmia and sometimes with anæmia. Small or large areas of softening or hæmorrhage with thrombosis may be present.

In the lungs giant cells in the pulmonary capillaries may be found and have been identified with masses of syncitium. They can also be found in pregnant women dead of other diseases.

Œdema of the lungs is a common complication.

Symptoms and Physical Signs.

Before enumerating and discussing the symptoms and physical signs I would like to mention that the urine of a considerable number of pregnant women contains albumin—10% to 15% of all pregnat women. The admission ward list at the Royal Hospital for Women shows a much higher figure, but catheter specimens of urine were not taken. This albuminuria is symptomless and occurs particularly at the end of pregnancy and during labour. It disappears on delivery. It has been suggested that it is due to renal congestion and is comparable to the albuminuria of athletes in training.

There are three stages of severity of the toxæmia:

(i.) Earliest stage (albuminuria of pregnancy).
(ii.) Second stage (preeclamptic symptoms).
(iii.) Third stage (eclamptic convulsions and coma).

The Earliest Stage: Albuminuria of Pregnancy.

The presence of albumin in the urine is the characteristic feature. It is present in the vast majority of cases. Rarely is it absent. The amount varies and seems to correspond roughly to the severity of the symptoms, although there are exceptions to this. The amount of urine excreted is generally diminished in quantity. Occasionally there is suppression. Casts are generally present and sometimes blood.

Œdema is present in a considerable proportion of cases. The extent of this varies. Sometimes there is general anasarca and other times only slight œdema of the ankles. The common sites are the ankles, face and lower part of the back. Even when actual pitting is absent, there seems to be a superficial fullness which suggests an œdema of the deeper tissues. It is fitting to mention again a type of patient which seems to me to be characteristic of this toxæmia. We see a young expectant mother, whose age is usually sixteen to nineteen years, with fair or auburn complexion, fat and with a bloated face, with puffy eyes and thick neck. A large percentage of these patients conform to this type.

Headache is not always present and is usually frontal in character. It may be very severe. Vomiting may occur even apart from the ingestion of food. Dizziness may be present. The temperature is normal. The pulse rate is normal. The tongue is moist and furred, breath foul, heavy and non-urinous. Some claim that there is a distinctive odour. Jaundice is sometimes present. Constipation is usually an accompaniment. The blood pressure is usually raised to 130 or 160 millimetres of mercury. (The blood pressure will be discussed in detail later.) There is an accentuated aortic second sound.

In regard to the respiratory system when œdema is a prominent feature, there are usually some fine crepitations to be heard at both lung bases. Œdema of the lungs is very rare, but has been reported.

The blood urea estimations reveal a normal level of twenty to thirty-five milligrammes per one hundred cubic centimetres of blood.

The Second or Preeclamptic Stage.

Preeclamptic symptoms are generally sudden in onset. They may become severe in a very short time, but they may take several hours to become severe. Other symptoms are aggravated.

Vomiting becomes frequent. Headache is present practically always and is generally frontal. I have noted a severe pain occurring at the nape of the neck, which almost surely ushers in convulsions.

Visual symptoms are stated to be not very common, but in my experience the opposite has been the case. They are fairly frequent. Dimness of vision, photophobia, diplopia, spots, flashes of light and temporary blindness are found.

I well remember one young patient, who developed flashes of light and blindness, while she was being delivered. Her urine which previously had been free from albumin, became solid on boiling. Her visual symptoms were the first and practically the only indications of the toxæmia.

It has been suggested that the visual symptoms are due to an œdema of the higher visual centres.

An hemianiopsia may develope during the puerperium. Irritability sometimes is noticed. There is a distaste for examination. The patient sometimes becomes morose. Dizziness is present.

Epigastric pain is usually severe and generally ushers in convulsions. It occurs in about 60% of cases. It is probably due to focal hæmorrhages under the liver capsule. This has been supported by *post mortem* examination. Some of these hæmorrhages are fairly extensive.

The temperature is usually normal; the pulse rate is definitely raised (90 to 120 per minute).

The amount of urine is diminished. Albumin is generally increased in amount. Blood may appear.

There is a definite rise of blood pressure to 160 or 200 millimetres of mercury. This rise may be sudden or more gradual. It is probably the first indication of an aggravation of the toxæmia.

The urea content of the blood is normal.

All these widely dispersed symptoms indicate a sudden aggravation of the toxæmia.

The Third Stage: Eclamptic Convulsions.

Eclamptic convulsions present three distinct phenomena: (i.) Preliminary twitching which lasts from five to fifteen seconds. This is accompanied by conjugate deviation of the eyes. (ii.) Tonic convulsions with deep cyanosis which lasts one to two seconds. (iii.) Clonic convulsions with increased cyanosis and profuse secretion of saliva. Its duration is generally from eighty to one hundred and eighty seconds.

After a period of coma consciousness is regained. The period of coma gradually increases after successive convulsions. It eventually becomes continuous and has been known to last for three days. Occasionally coma may supervene without antecedent phenomena and the *post mortem* findings in these cases consist of typical hepatic lesions.

Convulsions may occur before, during or after delivery. I believe that the convulsions are *ante partum* in the greatest percentage of cases. Labour frequently begins shortly after the onset of convulsions and this is one argument in favour of the conservative method of treatment.

The temperature is normal at the outset. It rises with the increasing severity of the condition and the frequency of the convulsions. It may be very high, particularly just before death. It is said to be raised during the convulsions.

The pulse rate is raised and increases with the severity of the condition. It is raised during the convulsions and remains somewhat elevated for some days after the final seizure.

There is a constant rise of blood pressure to 160 or 200 millimetres of mercury. It is raised during the convulsion. The aortic sound is definitely accentuated.

The tongue is swollen and furred and may be lacerated from being bitten. A heavy, foul, non-

urinous odour is said to be characteristic of the breath. There is tenderness and rigidity in the region of the right hypochondrium. This is not uncommon.

Respiration is stertorous. Adventitious signs are frequently present at the bases of the lungs. Œdema of the larynx has occurred.

The blood urea content is normal.

The amount of urine is reduced. There may even be complete suppression. Albumin is present and generally in large quantities. Occasionally albumin has been absent just prior to the onset of convulsions, but it is always present once convulsions have been established. Blood is frequently present in the urine, sometimes as hæmaturia and sometimes as meth-hæmoglobinuria. Diacetic acid and acetone are occasionally to be found. Hyaline and granular casts are the rule. The ammonia coefficient is generally high. The significance of this is not clear and does not justify the interpretation that there is any great retention of nitrogenous material in these subjects.

A considerable increase in the sugar content of the blood has been reported and this has been attributed to increased muscular activity incident to the convulsions.

The immediate cause of death is usually œdema of the lungs or cerebral hæmorrhage, but if the fatal issue be delayed for several days it is usually due to pneumonia or puerperal infection.

Jaundice occasionally supervenes and is of grave prognostic import.

In the differential diagnosis of albuminuria of pregnancy one has to eliminate the possibility of epilepsy, hysteria, cerebral hæmorrhage, uræmia, poisoning from strychnine or phosphorus. Most of these conditions offer little difficulty and I do not propose to consider them.

The Blood Pressure.

The study of the blood pressure in this type of toxæmia and in pregnancy complicated by nephritic conditions has come into prominence during the past few years. The readings and especially regularly charted readings afford valuable information with regard to diagnosis, prognosis and treatment.

The systolic blood pressure is of most value.

The Value of Blood Pressure Readings in Different Conditions.

As has been pointed out by various observers and especially in Australia by Professor Windeyer, the charting of the blood pressure helps in diagnosis and especially in dividing this type of toxæmia into the groups that have been previously described. Speaking generally the blood pressure figures in the various groups are as follows:

(i.) Uncomplicated toxæmia exhibits a blood pressure of 130 to 150 millimetres of mercury which rises considerably in the preeclamptic and eclamptic stages, even as high as 200 millimetres of mercury.

(ii.) Nephritic toxæmia is characterized by a blood pressure of 150 to 200 millimetres of mercury with corresponding elevations as in the former group, but the elevations are smaller.

(iii.) Uræmia is accompanied by a blood pressure of over 200 millimetres of mercury.

The figures are fairly constant and they are of great assistance. There are, however, three points that minimize its value.

In the first place the patient may come under observation for the first time in convulsions and naturally the blood pressure is high in all three groups. Sometimes the blood pressure may be higher in Group (i.) than in Group (ii.) in the convulsive stage. In the list of histories attached to this paper there will be noticed figures bearing out this statement. In one subject with typical signs and history of nephritic toxæmia and a blood urea content of 50 milligrammes per one hundred cubic centimetres of blood, the maximum blood pressure was 160 millimetres. In another, a typical toxæmia, with a blood urea content of 24 milligrammes per one hundred cubic centimetres of blood, the maximum blood pressure was 180 millimetres.

In the second place it is an established fact that accidental hæmorrhage is likely to occur in this toxæmia. Of course minor hæmorrhages will temporarily lower the blood pressure, but a severe *ante, intra* or *post partum* hæmorrhage may lower the blood pressure for some considerable time. In one of the cases recorded the patient had a severe accidental hæmorrhage. Unfortunately the blood pressure was not recorded, but it was obviously lower than normal (the pulse being very "soft"). The patient died of uræmia with a blood urea content of 250 milligrammes per one hundred cubic centimetres of blood.

In the third place when heart failure is a complication the blood pressure is low.

These illustrations are cited only to show that occasionally the blood pressure cannot be relied upon for guidance.

I believe that the greatest value of the blood pressure readings is in the diagnosis of the onset of the preeclamptic and eclamptic stages, especially when they are recorded every two hours or even more frequently. There are some patients who will develope preeclamptic symptoms in spite of treatment. I have seen a considerable number of these and have been struck by the constant and early elevation of the blood pressure. In many instances this elevation was present before the usual symptoms appeared. If the blood pressure were taken very frequently (for example, every half hour) in these patients who are under treatment, I believe it would be the first sign of an aggravation of the toxæmic condition.

Also eclamptic convulsions occasionally occur without any warning symptoms. Permit me to mention one instance.

A woman was admitted with revealed accidental hæmorrhage. The usual treatment was carried out (vaginal plugging *et cetera*) and labour had commenced. The urine revealed a cloud of albumin on boiling. The blood pres-

sure was being recorded every half hour and the readings varied between 110 and 120 millimetres. It rose to 140 millimetres and a few minutes later the patient had an eclamptic convulsion. There were no preeclamptic symptoms. The rise in blood pressure was the only warning of the onset of serious symptoms. In the absence of the usual symptoms the seriousness of the rise in blood pressure was not recognized, otherwise an attempt would have been made to stave off the onset of convulsions.

Again the diagnostic value of the blood pressure is demonstrated in those instances in which convulsions develope for the first time after delivery or in which they continue after delivery. The blood pressure falls somewhat after delivery and the average reading is considerably lower. A rise of blood pressure at this stage will herald convulsions or if the average fall does not take place, a continuation of the convulsions may be expected. This point will be discussed again with regard to prognosis.

Blood Pressure Readings and Prognosis.

The chief prognostic value of blood pressure records lies in the regular charted readings during the puerperium. In the purely toxæmic type the blood pressure falls to normal in a few days and remains there. The normal figure (by the method of palpating the pulse at the wrist) to my mind is 105 to 115 millimetres of mercury. It should be noted here that any figures which have been given, have been taken by this method. The figures are lower than those obtained by the auscultatory method.

Any elevation of the blood pressure that persists, may be regarded as abnormal and indicating permanent damage to the kidneys. Thus we have definite grounds for giving an opinion about the future health of the patient and the advisableness of future pregnancies. In those instances in which the elevation persists, the albumin in the urine is very slow to disappear and may be present for months.

Occasionally the blood pressure falls to normal limits and still there are grounds for believing that the kidneys are permanently damaged. For example:

A patient with the fourth pregnancy and a history of previous "kidney trouble" was admitted on December 3, 1924. The urine on boiling contained solid albumin and the maximum blood pressure was 160 millimetres of mercury. General treatment, venesection and induction of labour by a de Ribes bag were carried out. The patient was delivered of a full-time, still-born child. The urea content of the blood was 50 milligrammes per one hundred cubic centimetres of blood. The patient was discharged on December 19, 1924. The urine was clear and the blood pressure was 110 millimetres. The blood urea content was again 50 milligrammes per one hundred cubic centimetres of blood (the patient was having a restricted protein diet). Here the blood pressure on discharge was normal, but the patient had chronic nephritis.

Thus we can definitely state the following:

(i.) A guarded prognosis should be given if the blood pressure remains elevated.

(ii.) The fall of the blood pressure to normal does not necessarily mean that a good prognosis may be given.

Again, after delivery the accoucheur will always be asked if there will be any more convulsions or if the patient will remain well. The blood pressure readings aid in answering these questions. A persistence of an average high blood pressure or a sudden elevation after the usual temporary fall are indications of further convulsions. Cessation of convulsions nearly always coincides with a considerable average fall of blood pressure or even a sudden fall to near the normal limits.

Let it be clearly understood that the blood pressure *per se* may be of no use and even misleading. A correct interpretation can be obtained only when it is considered with the other available evidence.

Blood Pressure Readings and Treatment.

A steady rise of blood pressure in spite of preventive treatment is to my mind a sure indication for terminating pregnancy.

Also, as has been pointed out previously, a sudden rise of blood pressure is a danger signal on which we must act promptly. It may indicate the exhibition of "Veratrone" or morphine and perhaps immediate induction of labour.

Again, should it be decided to use "Veratrone" in the preeclamptic or eclamptic stages, the taking of blood pressure readings every five or ten minutes is absolutely essential. The frequent readings afford us an idea of the efficacy of the "Veratrone" treatment and the blood pressure records cannot be dispensed with, if this method of treatment is to be intelligently employed.

The Estimation of the Urea Content of the Blood.

The blood urea content was estimated in the case of forty patients including those suffering from albuminuria of pregnancy in all three stages and gravid women with chronic interstitial nephritis and uræmia. The conclusions drawn as to the value of this test as an aid to diagnosis and prognosis have been made from figures in these cases. The estimations were made by biochemists at the Royal Prince Alfred and Sydney Hospitals whom I thank for their kindness. The patients were all having a diminished protein diet.

The urea concentration test was not performed, but it is stated (de Wesselow and Wyatt) that it yields corresponding results. Circumstances would not permit the carrying out of this test.

The normal blood urea content has been given at a figure varying between twenty and thirty milligrammes per one hundred cubic centimetres of blood. Evidence has been brought forward to support the fact that there is a low nitrogenous metabolism in the latter months of pregnancy. One effect of this is that urea is formed in relatively small amounts. Thus in the later months of pregnancy the quantity of urea present in the blood is usually less than twenty milligrammes per one hundred cubic centimetres of blood (de Wesselow and Wyatt).

Value of Blood Urea Estimations in Different Conditions.

By the estimation of the blood urea content we can definitely distinguish toxæmia from uræmia and a true toxæmia from one superimposed on a pre-

existing chronic nephritis. The figures that have been obtained are as follows:

(i.) True toxæmia: twenty to forty milligrammes per one hundred cubic centimetres of blood.

(ii.) Nephritic toxæmia: fifty to one hundred milligrammes per one hundred cubic centimetres of blood.

(iii.) Uræmia: two hundred and fifty milligrammes per one hundred cubic centimetres of blood.

In the first group it will be seen that the blood urea content is within normal limits. The blood was withdrawn from patients during the first, second and third stages of the toxæmia and also from some during convalescence. In many subjects two figures were obtained: (i.) during the convulsive stage and (ii.) on recovery. These figures were practically the same and in many actually the same. To give an example:

Mrs. B. was admitted on October 3, 1924. The blood urea content was thirty milligrammes during convulsions and was twenty-five milligrammes in convalescence on October 19, 1924.

A series of estimations were not obtained in each case for obstetric and other reasons, but I do not think that the blood urea content varies to any great extent during the course of the illness.

In the second group there is a definite rise in the urea content of the blood.

In the third group the figures correspond to that of uræmia uncomplicated by pregnancy.

Blood Urea Estimations and Ætiology.

The results from the ætiological point of view are inconclusive. They prove that this toxæmia is not a true uræmia, although it is clinically similar in some ways to that disease. This view has been entertained for many years.

Blood Urea Estimations and Diagnosis.

Some value may be claimed for the test in diagnosis. Figures have been quoted dividing cases with very similar clinical pictures into three groups. These groups have been described for many years, but here is a definite basis on which a diagnosis can be given. As has been stated previously a true toxæmia (that is albuminuria, preeclamptic state and eclamptic convulsions) shows a figure which is normal or subnormal, a nephritic toxæmia a definitely raised figure and a uræmia a figure similar to that of uræmia apart from pregnancy.

It may be argued that blood urea estimations are superfluous and that the diagnosis can be established by other means, for example, previous history, clinical picture *et cetera*. This is so in many instances, but there are some cases in which the damage to the kidneys cannot be definitely estimated by the above means. This is a vital and necessary detail to know, especially with regard to prognosis and to the question of future pregnancy. The blood pressure records are a valuable aid, but they are not always a reliable guide.

To give the blood urea figures their due significance the exact diet of the patients must always be borne in mind.

Blood Urea Estimations and Treatment.

With regard to treatment it is sometimes difficult to decide whether induction of abortion or of premature labour should be carried out and there will be a difference of opinion as to the extent of the kidney damage already present. With an estimation of the blood urea a valuable guide is placed at our disposal. I should say that a figure more than fifty milligrammes per one hundred cubic centimetres of blood is a definite indication for the termination of pregnancy.

Although improvement seems to follow the termination of pregnancy in many cases of nephritic toxæmia, we must bear in mind that others become progressively worse and may even die rapidly of uræmia. I venture to suggest that this latter happening is a tragedy that might be easily averted by the timely estimation of the urea content of the blood. It is the risk of this happening that we must always bear in mind. The reason for it probably lies in the fact that a large amount of protein is freed into the blood by autolysis from the uterus during its involution. This accumulation is probably accentuated by the fact that no milk supply may be established and this avenue for the using up of the excess protein is lost. This often happens because the infant is generally stillborn. This accumulation of protein in the blood proves too much for the already damaged kidneys and uræmia results. Thus, to my mind, blood urea estimations are not only important but necessary.

Blood Urea Estimations and Prognosis.

In regard to the immediate prognosis the blood urea estimation is of little aid in helping one to give an opinion about the immediate outcome of cases belonging to the first two groups. Other features help in this respect, particularly certain clinical signs. With regard to uræmia a very high figure naturally is of grave prognostic import.

In regard to ultimate prognosis valuable information is afforded, especially with regard to the future health of the patient and also with regard to future pregnancy.

In a true toxæmia the figure is normal and the patient should enjoy good health and there is nothing against future pregnancies. In a subject showing a moderately high figure (over fifty milligrammes) the prognosis as to future health should be guarded and the risk of further damage to the kidneys by future pregnancy becomes very real and advice against pregnancy should be given. In those showing high figures the prognosis must be gloomy.

Thus we have definite guides for opinions to be given with regard to two important questions which are sure to be asked by the patient or by the patient's relatives.

COMPLICATIONS.

The following complications need to be considered.

Œdema of the lungs is frequent and is the cause of a considerable percentage of deaths. It may be due to the excessive exhibition of saline transfusion

or infusion. It may be caused by the use of croton oil or pilocarpine. It may be due to cardiac failure. It is liable to follow ether anæsthesia, particularly a prolonged anæsthesia. Bleeding, purging and stimulants are of little avail. It is claimed that atropine is of service.

Œdema of the larynx is rare, but has been known to follow the use of croton oil and pilocarpine.

Broncho-pneumonia may develope during convalescence. It is generally due to the aspiration of saliva or of sloughing matter from tongue injuries.

Injuries to the tongue may be very serious and severe secondary hæmorrhage may occur. Prevention of this is important.

Contusions, injuries and burns are rare. It should be noted that these subjects seem to be particularly liable to burning.

Cerebral hæmorrhage is a fairly frequent cause of death. Large hæmorrhages are rare, but multiple small hæmorrhages may be more common.

Albuminuric retinitis and suppression of urine also occur. Uræmia is very rare. An estimation of the blood urea content would settle the diagnosis.

Coma and acute yellow atrophy of the liver have occurred.

Extensive intraperitoneal hæmorrhage has been reported and occurred from the rupture of a subcapsular hepatic hæmatoma.

Accidental hæmorrhage is another complication.

Puerperal infection seems particularly liable to occur, probably on account of the exhausted condition of the patients.

The causes of premature labour are: (i.) Accidental hæmorrhage; (ii.) placental infarction; (iii.) others apparently due to the toxæmia. Stillbirths are frequent from the above causes and prematurity.

PROGNOSIS.

Maternal Prognosis.

The death rates of mothers in different clinics vary widely. It may be stated to be from 5% to 20%. It is said to be worse as a rule in those starting before delivery than in those starting during or after delivery. The most severe cases of all are seen in the puerperium. Bad prognostic signs are: (i.) When convulsions supervene on a chronic nephritis. This is probably the reason why they are stated to be worse in *multiparæ*. (ii.) The early advent of coma and the rapid succession of convulsions. (iii.) Suppression of urine. (iv.) Hyperpyrexia. (v.) The absence of œdema. (vi.) The advent of œdema of the lungs and cardiac failure. (vii.) Exceedingly rapid pulse rate with high tension and also a rapid thready pulse. (viii.) The advent of acute yellow atrophy is a very grave sign.

The ultimate prognosis to the mother has been discussed previously. It only needs to be said here that the outlook, both with regard to future health and future pregnancies, is good. Chronic renal disease rarely results and eclamptic convulsions rarely recur. It is this latter fact that suggests the possibility that an immunity has been conferred on the patient.

Fœtal Prognosis.

The mortality as far as the fœtus is concerned is very high. It is probably due to: (i.) The toxæmia; (ii.) prematurity; (iii.) placental infarction; (iv.) injury from operative interference.

The presence of albumin in the infant's urine and the occurrence of convulsions after birth, apart from the finding of cerebral lesions, suggest that the infant suffers from the toxæmia in the same way that the mother does.

TREATMENT.

I realize that most of what is to be written down under the heading of treatment can be found in any book of midwifery. I am setting it out in detail because here and there are certain points of interest on which I would like to record my own experience and views.

There is no doubt that no single line of treatment is the correct one in the eclamptic stage of this toxæmia. Each case must be dealt with on its own merits and there is no condition in which previous experience is of more value in the treatment of a given case. I would like to make a plea for conservatism of treatment and the minimum of obstetric interference in the third (eclamptic) stage. The mere fact that a patient with eclamptic convulsions who has had treatment by the conservative method persevered with, may recover and have a normal full-time confinement, should make one support this method of treatment. This does happen and would occur more frequently but for some method of obstetric interference which is more frequently resorted to.

Treatment of the First Stage: Albuminuria.

Prevention consists in: (i.) Frequent examinations of the urine; (ii.) dieting during the last few weeks of pregnancy; (iii.) regulation of the bowels.

Actual treatment aims at diminishing the body metabolism and assisting the elimination of toxins.

Rest in bed with blankets and hot bottles are necessary to promote diaphoresis. Hot air baths are stated to be weakening. Pilocarpine is to be avoided on account of the risk of œdema of the lungs.

In regard to diet water alone is given till the symptoms disappear and the albumin is appreciably diminished or absent from the urine. The patient should then be allowed only a very restricted diet. During labour owing to the risk of a sudden flooding of toxins into the system all food should be discontinued until twenty-four hours after delivery.

For purposes of purgation magnesium sulphate or *pulvis jalapæ compositus* are used with or without enemata.

Diuresis may be promoted by plenty of water or *potus imperialis* and hot kidney packs frequently applied to the loins.

Surgical treatment consists of a puncture of the labia or of the legs for excessive œdema. It is rarely indicated.

The indication for induction of premature labour is an increase in the toxæmic symptoms in spite of treatment. It is usually carried out by Kraus's bougie method or by the insertion of a Champetier de Ribes bag. Gordon Ley happily states:

> It is extraordinary with what frequency the uterus agrees with the obstetrician and itself terminates pregnancy at the time when he has decided that induction has been necessary.

I have been struck by the large proportion of cases which do not respond to treatment and in whom induction has to be resorted to. In the appended list there are twenty-three cases of albuminuria of pregnancy (first stage). In seven of these pregnancy was terminated (five by induction and two by Cæsarean section). Taking the figure at five we see that in 22% of cases pregnancy had to be terminated. Thus it will be seen that too optimistic a view must not be taken about the efficacy of preventive treatment apart from the termination of pregnancy. I do not agree with those who state that eclamptic convulsions can be prevented in every case.

The fact that in the majority of cases convulsions do not supervene forces one to the conclusion that antenatal care and preventive treatment are very real and important agents in the life saving to the mother and child. Also the value of this treatment is enhanced by the fact that when convulsions do occur in spite of it, they are less severe and less frequent.

Treatment of the Second or Preeclamptic Stage.

In regard to the treatment of the second (preeclamptic) stage I will confine my remarks to those instances in which preeclamptic symptoms develope whilst treatment is being carried out.

There is no doubt that the onset of these symptoms is a definite indication for the immediate induction of labour. In practically every instance the fœtus is at a viable age and there is no need for delay on that score. Thus the main line of treatment is induction of premature labour as soon as possible, but some time must elapse during the preparation for this operation. It may be many hours (perhaps relatives have to be sent for). There may be many causes for delay. It is in this interval that something must be done for the patient. At any moment convulsions may start. These must be prevented.

Sedative treatment is recommended by most authorities, that is morphine hypodermically in doses of 0·015 to 0·03 gramme (one-quarter to one-half grain) which may be repeated. Chloral hydrate and tincture of opium are also recommended. There is no doubt that morphine controls the symptoms in most of the cases, but it has two disadvantages. It is not always effective in preventing convulsions. It has a bad effect on the child which, although slight at term, is considerable on a premature infant. Add to this the effect of one or two inductions of anæsthesia and the chances of a premature child being still-born are considerable.

I believe a better method of controlling these symptoms is to be found in the use of "Veratrone" in doses of 0·3 to 0·42 mils (five to seven minims). I have had signal opportunities for noting the effect of this drug and I submit that in controlling the preeclamptic symptoms it has fulfilled every requirement.

"Veratrone" in Treatment.

It is opportune to say here a few words about "Veratrone"—a drug that has been widely praised and widely maligned. As I have never seen *veratrum viride* used, I will confine my remarks to a trade preparation of that drug, namely "Veratrone." There is one essential condition in its use. There must be a competent person continually in attendance to take frequent readings of the pulse rate and blood pressure. There has been a considerable amount of discussion as to the efficacy of this drug, some observers absolutely condemning it and others going to the other extreme. To my mind, there are certain occasions when "Veratrone" becomes a useful adjunct to the treatment of this toxæmia.

In the eclamptic stage its effect varies with different cases. If a definite reaction occur, "Veratrone" should be persevered with. By a definite reaction is meant a fall of blood pressure to normal or below normal (90 to 110 millimetres of mercury) and a fall of the pulse rate to eighty per minute or less.

The efficiency of "Veratrone" depends on its capacity to lower the blood pressure. Should this result, the charting of the pulse and blood pressure will give an indication for its further use. The initial dose is usually 0·3 to 0·42 mils (five to seven minims) and is given hypodermically. Several further injections are generally required. These are smaller (0·12 to 0·18 mils or two to three minims) and may be repeated every half hour. This will take the patient over an interval until the bowels are acting freely and this seems to be the only time that "Veratrone" will be of assistance (Professor Windeyer). It is well known that once the bowels are acting freely the tendency is in the majority of cases for the convulsions to subside.

Again I think that "Veratrone" can be combined very well with sedatives such as morphine, always assuming that there is a definite reaction. If it can be used alone, it is preferable to morphine, but these instances are rare.

A great disadvantage of its use is that it will not quieten the patient, although it controls the convulsions. Two attendants are thus needed at the patient's bedside all the time.

It has little effect on those toxæmias which have been superimposed on a chronic nephritis.

The conclusions about its value in the eclamptic stage are: (i.) "Veratrone" controls the convulsions in many instances. (ii.) It is preferable for it to be given in combination with morphine. The dose of morphine is then considerably smaller than if it were used alone.

In the first and second (preeclamptic) stage "Veratrone" is to my mind of most use. The sudden onset of preeclamptic symptoms in patients who

are receiving dietetic and other treatment, can be effectively controlled by the exhibition of "Veratrone." I have repeatedly noted the beneficial effect of "Veratrone" (0·3 mils) with perhaps one or two further doses of 0·18 mils. Occasionally one injection was sufficient and there was no return of symptoms. In the case records there will be noted several instances where preeclamptic symptoms were controlled by "Veratrone" alone.

Treatment of the Third or Eclamptic Stage.

In regard to prevention of the third (eclamptic) stage two points require emphasis.

In the first place routine examination of the urine during pregnancy is of the first importance. The percentage of eclampsia that can be prevented, has been given at very variable figures. Some say that 90% can be prevented, others give as low as 10%. We must remember two things in estimating a figure like this. Firstly, some cases of untreated toxæmia do not end in convulsions and secondly, a history of a few hours' duration is sometimes the case. Thus an accurate figure cannot be obtained. Probably 66% of cases are preventible.

In the second place the recognition of pre-eclamptic symptoms is important. In regard to actual treatment the patient must be prevented from injuring herself and especially her tongue.

For control of the convulsions I have used chloroform repeatedly and have noted no ill effects. It is stated to intensify any damage that may be present in the liver. Chloroform was used sparingly. It is remarkable how little is necessary. Free use of chloroform I believe to be a dangerous practice. Œdema of the lungs is a very real danger and for this reason I think ether is best avoided. Chloral hydrate and bromides by the mouth or rectum have been advocated. In the few cases in which I have seen them used, they were ineffective. Tincture of opium is used in some parts of the world. Morphine in 0·015 to 0·03 gramme (one-quarter to one-half grain) doses is very useful and certainly controls the convulsions. As much as 0·18 gramme (three grains) has been recommended in twenty-four hours. "Veratrone" has been discussed previously. Suffice it to say that in combination with morphine it is very useful. Doses as large as one mil (fifteen minims) are recommended by some authors. I do not think the doses need be larger than 0·42 gramme (seven minims); in fact large doses are dangerous.

Elimination of the toxins by the bowels, kidneys and skin must be undertaken. For elimination by the bowels one or two enemata may be needed in the first place. *Pulvis jalapæ compositus* or magnesium sulphate are given or run into the stomach through a stomach tube. It is a good plan to wash out the stomach before giving these. Any manipulation of the patient, such as giving of enemata, catheterization, gastric lavage *et cetera*, should be carried out under light anæsthesia.

For elimination by the kidneys hot packs should be applied to the loins every half hour. Saline infusion into veins or by the bowels or under the skin should be avoided owing to the risk of œdema of the lungs. Diuretics should be avoided. Venesection has been warmly recommended. It is unwise to remove more than two hundred and forty to three hundred cubic centimetres (eight to ten ounces) of blood on account of the serious risk of an accidental *ante partum* or *post partum* hæmorrhage. The best indication for it is in a patient with *post partum* convulsions where there is deep cyanosis. It is an excellent manœuvre in such a case. A free flow of blood may be encouraged at the third stage.

For elimination by the skin hot blankets and hot bottles have probably the best effect. Stimulants such as digitalis, strychnine and alcohol may be needed. Lumbar puncture has been used with varying results. I have seen beneficial effects from its use, when there has been persistent headache during the early convalescence. The patient must be placed in an isolated shaded room. The patient should be kept on her side, so that the secretions of the mouth and upper respiratory passages may escape. Decapsulation of the kidneys has been advised for suppression of the urine.

In regard to obstetric treatment there can be no hard and fast rules.

For the majority of cases the following is sound advice: If the patient is in labour, treat as an ordinary case and avoid forcible delivery.

Induction of labour is said to be advisable in certain cases. I cannot subscribe to this; it is probably rarely necessary.

Cæsarean section is favoured in certain centres. To my mind it is preferable to induction of labour. The best figures obtained from this method are 10% maternal mortality. Better figures have been obtained by conservative methods. Cæsarean section in all cases is not indicated. I think it should be performed for those patients who have not started in labour and in whom there has been no rapid improvement by conservative methods. It should also be done when there are indications apart from toxæmia. Williams advises quick delivery according to the state of the *os cervici*:

(1) Forceps—*os* at full dilatation.

(2) Dilatation and forceps—*os* easily dilatable to full dilatation.

(3) Cæsarean section—*os* undilated.

The puerperium must be carefully looked after. The patients are prone to heart failure, pneumonia and septic infection. These are treated on ordinary lines once they arise.

Sedatives (paraldehyde or morphine) may be necessary for some days, especially if there be signs of cerebral irritation.

During the convalescence there is usually dulness of intellect and slow cerebration for a day or so. In some patients there is more or less insanity. Most of these recover.

The infant sometimes has convulsions. Small doses of chloral hydrate and bromide should be given. The child should not be allowed to suckle for a few days.

NEPHRITIC TOXÆMIA.

Before closing this paper I do not think it would be out of place to make a brief reference to nephritic toxæmia.

Definition.

Toxæmic symptoms supervene in a gravid woman who is suffering from chronic interstitial nephritis.

Ætiology.

It occurs more frequently than one is led to believe. It is present mainly in *multiparæ*. Scarlet fever and chorea are definite causal agents, also previous toxæmia of pregnancy. Abnormalities and other lesions of the kidneys predispose to it.

Pathology.

The pathology is the same as that of chronic interstitial nephritis uncomplicated by pregnancy. Both old and recent infarcts are frequently found in the placenta.

Prognosis.

In regard to prognosis many patients go through pregnancy without serious consequences, but there is a grave risk of uræmic coma and death. This is especially likely to occur during the puerperium.

Eclamptic convulsions and other severe toxæmic manifestations occur with considerable frequency. When convulsions occur, there is a high maternal death rate. It is a common cause of repeated abortion and premature labour. The child very frequently dies.

Symptoms and Clinical Signs.

There is general malaise, headache and perhaps slight œdema. Sometimes ocular symptoms are present.

The blood pressure is high and the vessel walls are thickened. There are signs of cardiac hypertrophy. Albuminuric retinitis is present in a percentage of cases. The urine shows all the characteristics of a chronic nephritis. The blood urea content is definitely raised.

Differential Diagnosis from Albuminuria of Pregnancy.

1. The daily amount of urine may be unchanged or increased and there is low specific gravity.
2. Blood pressure is on an average definitely higher and persists above the normal for a considerable period after the confinement.
3. Blood urea content is definitely raised.
4. There may be a history of fevers or previous toxæmia.
5. Retinal hæmorrhages and albuminuric retinitis may be present (only œdema has been noted in albuminuria of pregnancy).
6. Persistence of albumin and casts in the urine for months.

Treatment.

The preventive and general treatment is the same as that prescribed for albuminuria of pregnancy.

For general guidance as to the induction of premature labour, the following is a good rule. Early in pregnancy induction of labour is needed if there be no rapid improvement under treatment; late in pregnancy try to get a viable child.

The following are the indications for the induction of premature labour:

1. Blood urea content of more than 50 milligrammes per one hundred cubic centimetres of blood.
2. Increase in the toxæmic symptoms in spite of treatment.
3. The presence of albuminuric retinitis.
4. The advent of eclamptic convulsions.
5. When the child is viable immediate induction is indicated.

CONCLUSION.

I realize that much of what I have written can be found in textbooks and articles on this subject. I have read a considerable amount of literature and a list of these authors is appended at the end of the paper. All through the paper I have tried to give my personal views which have been formed from such experience that I have been fortunate enough to have obtained. I think that I have raised several points of interest and I have tried to make no conclusions without sufficient data for their support.

ACKNOWLEDGMENT.

I would like to express my thanks to the honorary medical staff of the Royal Hospital for Women for their kindness in allowing me to use the material relating to the subject matter of this paper.

BIBLIOGRAPHY.

J. M. Munro Kerr: "Operative Midwifery."
J. B. de Lee: "Principles and Practice of Obstetrics."
J. Whitridge Williams: "Obstetrics."
R. W. Johnstone: "A Textbook of Midwifery."
Gordon Ley: "Practitioners' Encyclopedia of Midwifery and the Diseases of Women" (Fairbairn).
O. L. V. de Wesselow and J. M. Wyatt: "Modern Views of the Toxæmias of Pregnancy."
J. C. Windeyer: "Toxæmias of Pregnancy with an Analysis of One Hundred and Fifty-eight Cases of Eclampsia," *Transactions of the Australasian Medical Congress (British Medical Association)*, Supplement to THE MEDICAL JOURNAL OF AUSTRALIA, April 5, 1924, page 179.
J. C. Windeyer: "Blood Pressure Charts as an Aid to Diagnosis, Prognosis and Treatment in Albuminuria of Pregnancy and Eclampsia," THE MEDICAL JOURNAL OF AUSTRALIA, December 6, 1924, page 600.

CASE RECORDS.

Albuminuria of Pregnancy, Including Patients in the Preeclamptic Stage.

CASE I.—Mrs. B., aged twenty years, was pregnant for the first time. She gave a history of having had scarlet fever. Her urine contained "solid albumin," but no casts. It was clear on her discharge from hospital. Her systolic blood pressure was 95 to 100 millimetres of mercury. The blood urea was 33 milligrammes per hundred cubic centimetres of blood. She was delivered of a full time live infant.

CASE II.—Mrs. B., aged twenty-four years, was pregnant for the first time. Her urine contained "three-quarters solid albumin"; it was clear on her discharge from hospital. Her blood pressure was 120 millimetres of mercury. Her blood urea on September 11, 1924, was 33 milligrammes and on October 10, 1924, 28 milligrammes per hundred cubic centimetres. She was delivered of a full time live infant.

CASE III.—Mrs. W., aged twenty-two years, was pregnant for the first time. Her urine contained "solid albumin"; it was clear on her discharge. Her systolic blood pressure was 140 millimetres of mercury. On her discharge from hospital the figure was 110. Her blood urea was 32 milligrammes per hundred cubic centimetres, this estimation was made during the preeclamptic stage. She was delivered of a full time live infant. The preeclamptic symptom was controlled solely by "Veratrone."

CASE IV.—Mrs. B., aged twenty-two years, was pregnant for the first time. Her urine contained "solid albumin." On her discharge from hospital it was clear. The blood pressure was 140 to 160 millimetres of mercury. On her discharge from hospital the figure was 120. The blood urea on April 12, 1924, was 30 milligrammes and on April 25, 1924, it was 35 milligrammes per hundred cubic centimetres. Cæsarean section was performed for accidental, *ante partum* hæmorrhage and an eight months' live infant was born.

CASE V.—Mrs. R. M., aged twenty-four years, was pregnant for the second time. She was admitted to hospital on October 17, 1924, with a history of having had diphtheria and chorea. Her urine contained "one-quarter solid albumin." On her discharge from hospital it was clear. Her systolic blood pressure was 160 millimetres of mercury. The figure on her discharge from hospital was 120. Her blood urea on October 24, 1924, was 23 milligrammes per hundred cubic centimetres. She was delivered of a full time live infant.

CASE VI.—Mrs. B., aged twenty-three years, pregnant for the first time, was admitted to hospital on June 12, 1924. Her urine contained "three-quarters solid albumin." On her discharge from hospital on July 6, 1924, the amount of albumin was "one-quarter." Her systolic blood pressure on her admission was 150 millimetres of mercury and on her discharge was 120 millimetres. The blood urea was 28 milligrammes per hundred cubic centimetres on June 15, 1924. She was delivered of full time living twins.

CASE VII.—Mrs. B., aged twenty-four years, pregnant for the first time, was admitted to hospital on August 28, 1924. Her urine contained "solid albumin." On her discharge the urine contained merely a deposit of albumin. Her blood pressure on admission was 180 millimetres of mercury and on her discharge it was 100. The blood urea was 25 milligrammes per hundred cubic centimetres. Labour was induced and eight and a half months' living twins were born.

CASE VIII.—Mrs. B., aged twenty-four years, pregnant for the first time, was admitted on September 19, 1924. The urine on admission contained "half solid albumin"; it was clear on her discharge from hospital. The systolic blood pressure was 130 millimetres of mercury and on her discharge it was 100 millimetres. The blood urea was 26 milligrammes per hundred cubic centimetres. She was delivered of a full time living infant.

CASE IX.—Mrs. C., aged thirty-four years, pregnant for the fifth time, was admitted to hospital on March 29, 1924. The urine contained "solid albumin." On her discharge from hospital on July 19, it was clear. Her blood pressure was 160 millimetres of mercury. No blood urea estimation was made. Labour was induced and a full time living infant was born.

CASE X.—Mrs. C., aged twenty-two years, pregnant for the second time, was admitted on April 19, 1924. Her urine contained "three-quarters solid albumin." On her discharge from hospital on May 5, 1924, it was clear. The systolic blood pressure was 140 millimetres of mercury; on her discharge it was 110 millimetres. The blood urea was 24 milligrammes per hundred cubic centimetres. A full time living infant was born.

CASE XI.—Mrs. C., aged twenty-nine years, pregnant for the second time, was admitted on January 23, 1924. The urine contained "one-eighth solid albumin." On her discharge from hospital on February 21, 1924, it was clear. The systolic blood pressure was 140 millimetres of mercury and on her discharge 120 millimetres. No blood urea estimation was made. The patient was delivered of a seven and a half months' living infant.

CASE XII.—Mrs. G., aged twenty-four years, pregnant for the first time, was admitted to hospital on September 19, 1924. The urine contained "one-half solid albumin." On her discharge from hospital on October 3, 1924, it was clear. The systolic blood pressure was 140 millimetres of mercury and on her discharge 110 millimetres. The blood urea was 26 milligrammes per hundred cubic centimetres. She was delivered of a full time living infant.

CASE XIII.—Mrs. G., aged seventeen years, first pregnancy, was admitted on February 13, 1924. Her urine contained "solid albumin." On her discharge on March 5, 1924, it was clear. Her systolic blood pressure was 150 millimetres of mercury and on her discharge 110 millimetres. Her blood urea was 26 milligrammes per hundred cubic centimetres. Labour was induced and the patient was delivered of an eight and a half months' still-born infant.

CASE XIV.—Mrs. G., aged twenty-eight years, pregnant for the first time was admitted on May 2, 1924. The urine contained "half solid albumin." On her discharge on June 5, 1924, it was clear. The systolic blood pressure was 140 millimetres of mercury and on her discharge 110 millimetres. No blood urea estimation was made. The patient was delivered of eight months' living twins.

CASE XV.—Mrs. H., aged seventeen years, pregnant for the first time, was admitted on September 9, 1924. The urine contained "one-sixth solid albumin." On her discharge on October 3, 1924, it contained merely a trace of albumin. Her systolic blood pressure was 170 millimetres of mercury and on her discharge 90 millimetres. The blood urea was 26 milligrammes per hundred cubic centimetres on September 19, 1924. The patient was delivered of a full time living infant. Preeclamptic symptoms were controlled by "Veratrone."

CASE XVI.—Mrs. C., aged twenty-three years, pregnant for the first time, was admitted to hospital on August 7, 1924, with a previous history of scarlet fever and diphtheria. Her urine contained "solid albumin." On her discharge on August 20, 1924, it contained merely a trace of albumin. The systolic blood pressure was 180 millimetres of mercury and on her discharge 120 millimetres. The blood urea was 30 milligrammes per hundred cubic centimetres. On October 10 she was delivered of a full time living infant.

CASE XVII.—Mrs. M'O., aged twenty-two years, pregnant for the first time, was admitted on September 10, 1924. The urine contained "three-quarter solid albumin" and on her discharge merely a deposit of albumin. The systolic blood pressure was 190 millimetres of mercury and on her discharge from hospital 100 millimetres. The blood urea was 21 milligrammes per hundred cubic centimetres. She was delivered of an eight months' living infant. Preeclamptic symptoms were controlled by "Veratrone."

CASE XVIII.—Mrs. S., aged thirty-four years, pregnant for the first time, was admitted to hospital on July 3, 1924. Her urine contained "three-quarters solid albumin" and it was clear on her discharge. The systolic blood pressure was 150 millimetres of mercury and on discharge 110 millimetres. No blood urea estimation was made. She was delivered of full time living twins.

CASE XIX.—Mrs. W., aged thirty-one years, pregnant for the first time, was admitted on November 3, 1924. Her urine contained "three-quarters solid albumin" and on her discharge on November 28, 1924, was clear. The systolic blood pressure was 190 millimetres of mercury and on her discharge 130 millimetres. She was discharged from hospital unconfined and in the eighth month of pregnancy.

CASE XX.—Mrs M., aged twenty-one years, pregnant for the first time, was admitted to hospital on November 12, 1924. Her urine contained "three-quarters solid albumin"

and on her discharge on December 1, 1924, it was clear. Her systolic blood pressure was 190 millimetres of mercury and on her discharge 110 millimetres. The blood urea was 26 milligrammes at one examination and 24 milligrammes per hundred cubic centimetres on another occasion. Labour was induced and the patient was delivered of an eight and a half months' living child.

CASE XXI.—Mrs. C., aged seventeen, pregnant for the first time, was admitted to hospital on July 26, 1924. Her urine contained "three-quarters solid albumin" and on her discharge on August 26, 1924, it was clear. Her systolic blood pressure was 160 millimetres of mercury and on her discharge 110 millimetres. Her blood urea on August 12 was 20 milligrammes per hundred cubic centimetres. Labour was induced and the patient was delivered of an eight months' living child.

CASE XXII.—Mrs. D., aged thirty-nine years, pregnant for the first time, was admitted to hospital on November 12, 1924. Her urine contained "one-eighth solid albumin" and on her discharge from hospital it was clear. Her systolic blood pressure was 170 millimetres of mercury and on her discharge 110 millimetres. Her blood urea was 33 milligrammes per hundred cubic centimetres. Cæsarean section was performed on account of flat pelvis and a full term living child was born.

CASE XXIII.—Mrs. B., aged twenty-nine years, pregnant for the third time, was admitted on December 5, 1924, with a history of having had "similar trouble" with her first child. The urine contained "two-thirds solid albumin" and on her discharge from hospital on December 19, 1924, it was clear. The systolic blood pressure was 160 millimetres of mercury and on her discharge 110 millimetres. The blood urea was 30 milligrammes per hundred cubic centimetres. The patient was delivered of a full time living child.

CASE XXIV.—Mrs. E., aged thirty-five years, pregnant for the fourth time, was admitted on December 6, 1924, with a history of having had "kidney trouble" with each pregnancy. Her systolic blood pressure was 170 millimetres of mercury and on her discharge from hospital on December 19, 1924, was 110 millimetres. No blood urea estimation was made. Her urine contained "three-quarters solid albumin" and on her discharge it was clear. Labour was induced and the patient was delivered of a full term living child.

In the foregoing records no mention is made of the symptoms present. In all of the cases recorded all or some of the usual symptoms were present.

Again the blood pressure figure mentioned in the first instance is the maximum blood pressure of a series of readings.

A few points of interest may be noted in these twenty-four cases:

1. The persistent low blood urea figure.
2. The regular fall of the blood pressure.
3. The comparatively quick disappearance of the albumin from the urine.
4. Frequency of twins, four times in twenty-four cases.
5. The low infant death rate, one in twenty-four cases.
6. The fact that one subject was discharged unconfined.
7. The frequency of induction of labour or premature labour, six times in twenty-four cases.
8. The fact that preeclamptic symptoms were absolutely controlled in a few instances.
9. The preponderancy of women with first pregnancy.

Chronic Nephritis with Superimposed Toxæmic and Eclamptic Symptoms.

CASE I.—Mrs. McN., aged thirty-nine years, was pregnant for the sixth time. Her urine contained "one-eighth solid albumin." Her blood pressure was 160 millimetres of mercury. Her blood urea was 100 milligrammes per hundred cubic centimetres. She was delivered of a seven months' still-born infant and died of heart failure following the confinement. No estimation of blood urea content was made after delivery.

CASE II.—Mrs. H., aged forty-three years, pregnant for the eleventh time, was admitted on August 19, 1924. Her urine contained "one-quarter solid albumin" and was clear at the time of her discharge on September 3, 1924. Her systolic blood pressure was 240 millimetres of mercury and on discharge was 150 millimetres. She gave a history of previous "kidney trouble." No estimation of blood urea was made. Labour was induced and she was delivered of a seven months' still-born infant.

CASE III.—Mrs. M., aged thirty-six years, pregnant for the fifth time, was admitted with a history of previous "kidney trouble." She had had eclampsia with her fourth child. The urine contained "one-third solid albumin" and on discharge on December 11, 1924, contained merely a cloud of albumin. The systolic blood pressure was 200 millimetres of mercury and on discharge 120 millimetres. The blood urea was 80 milligrammes per hundred cubic centimetres. Labour was induced and the patient was delivered of a six and a half months' still-born infant. Lumbar puncture was performed on account of headache. Examination of the ocular fundi failed to reveal any abnormality. The patient had an eclamptic convulsion.

CASE IV.—Mrs. W., aged forty-three years, pregnant for the sixth time, was admitted on November 28, 1924, with a history of scarlet fever. Her urine contained "solid albumin" and on her discharge on December 12, 1924, merely a deposit of albumin was found. The systolic blood pressure was 180 millimetres of mercury and on her discharge 130. The blood urea was 60 milligrammes per hundred cubic centimetres. She was delivered of an eight and a half months' still-born infant and had eclamptic convulsions.

CASE V.—Mrs. D., aged thirty-four years, pregnant for the fourth time, was admitted on December 3, 1924, with a history of previous "kidney trouble." Her urine contained "solid albumin" and on her discharge on December 19, 1924, was clear. Her systolic blood pressure was 160 millimetres of mercury and on her discharge 110 millimetres. The blood urea was 50 milligrammes per hundred cubic centimetres. Labour was induced and the patient was delivered of a full time still-born infant. She had eclamptic fits.

The points of interest are:

1. The age of the patients (thirty-four to forty-three) and the fact that all are *multiparæ*.
2. All the infants are still-born. This fact is a very strong argument in favour of induction of abortion early in pregnancy when the blood urea estimation is fifty milligrammes or over.
3. The persistence of the blood pressure above normal.
4. The fact that induction was carried out in four instances.
5. The frequency with which eclamptic convulsions supervened.
6. The persistently raised blood urea content.

Uræmia.

CASE I.—Mrs. P., aged thirty-two, pregnant for the eighth time, was admitted on December 22, 1924. Her urine contained a cloud of albumin and blood. Her blood pressure was not estimated. The pulse was soft. She suffered

from profuse accidental hæmorrhage. She was delivered of a still-born child on the day of admission. She became comatose with suppression of urine on December 28, 1924. Her blood urea was 250 milligrammes per hundred cubic centimetres. The patient died on December 30, 1924.

Eclamptic Stage.

CASE I.—Mrs. B., aged twenty years, was pregnant for the first time. Her urine contained "solid albumin"; on her discharge it was clear. Her blood pressure was 180-160 millimetres of mercury and on her discharge 130-120 millimetres. Her blood urea was 30 milligrammes per hundred cubic centimetres during convulsions and 25 milligrammes on discharge sixteen days later. She was delivered of a seven months' still-born infant.

CASE II.—Mrs. B., aged twenty-four years, was pregnant for the first time. Her urine contained "solid albumin" and on her discharge it was clear. Her blood pressure was 160 millimetres of mercury and on her discharge 110 millimetres. Her blood urea was 28 milligrammes per hundred cubic centimetres during convulsions. She was delivered of an eight and a half months' living infant.

CASE III.—Mrs. B., aged eighteen years, was pregnant for the first time. Her urine contained "three-quarters solid albumin" and on her discharge a deposit of albumin was still present. Her blood pressure was 140 millimetres of mercury and on discharge 110 millimetres. Her blood urea was 20 milligrammes per 100 cubic centimetres during convulsions and stood at the same figures two months later. She was delivered by instruments of a still-born full time infant.

CASE IV.—Mrs. G., aged eighteen years, was pregnant for the first time. Her urine contained "solid albumin" and on her discharge a trace of albumin was present. Her blood pressure was 180 millimetres of mercury and on her discharge 110 millimetres. No estimation of blood urea was made. She was delivered of an eight and a half months' living infant. Delivery was instrumental on account of persistent occipito-posterior position. "Veratrone" and morphine were given to her simultaneously.

CASE V.—Mrs. H., aged twenty-two years, was pregnant for the first time. Her urine contained "three-quarters solid albumin" and on her discharge a deposit of albumin was still present. Her blood pressure was 200 millimetres of mercury and on her discharge 130 millimetres. No estimation of blood urea was made. She was delivered by instruments of an eight months' still-born infant. "Veratrone" was used alone.

CASE VI.—Mrs. M., was pregnant for the second time. Her urine contained "half solid albumin"; it was clear on her discharge. The blood pressure was 160 millimetres of mercury and on her discharge 110 millimetres. The blood urea was 40 milligrammes at the time of her admission and stood at the same level on her discharge fourteen days later. She was delivered of a seven and a half months' living child. She was given "Veratrone" and morphine and was subjected to venesection.

CASE VII.—Mrs. M., aged twenty-six years, was pregnant for the second time. Her urine contained "one-quarter solid albumin"; on her discharge it was clear. Her blood pressure was 150 millimetres of mercury and on her discharge 110 millimetres. Her blood urea was 35 milligrammes per hundred cubic centimetres. She was delivered of a seven and a half months' still-born infant. "Veratrone" and morphine were used.

CASE VIII.—Mrs. S., aged twenty-three years, was pregnant for the first time. Her urine contained "solid albumin" and on her discharge it was clear. Her blood pressure was 160 millimetres of mercury and on her discharge 110 millimetres. Her blood urea was 26 milligrammes per hundred cubic centimetres. She was delivered by instruments of an eight and a half months' still-born infant. Morphine and "Veratrone" were used.

CASE IX.—Mrs. S., aged twenty-one years, was pregnant for the first time. Her urine contained "one-quarter solid albumin." Her blood pressure was 140 millimetres of mercury and on her discharge 110 millimetres. Her blood urea was 30 milligrammes during convulsions and 24 milligrammes on discharge twenty-one days later. She was delivered of a full time living child. Her eclampsia was of the *intra partum* type. Convulsions occurred between the second and third stages of labour. Morphine was used.

CASE X.—Mrs. S., aged thirty-four years, was pregnant for the fifth time. Her urine contained "one-third solid albumin" and on her discharge a cloud of albumin was still present. Her blood pressure was 150 millimetres of mercury and on her discharge 120 millimetres. Her blood urea was 30 milligrammes per hundred cubic centimetres, the estimation being made twenty-one days after the occurrence of convulsions. She was delivered of full time living twins. Her eclampsia was of the *post partum* type.

CASE XI.—Mrs. W., aged thirty years, was pregnant for the third time. Her urine contained "one-quarter solid albumin" and on her discharge a trace was still present. Her blood pressure was 120 millimetres of mercury and on her discharge 110 millimetres. No estimation of blood urea was made. She was delivered of a living full time child. Her eclampsia was of the *post partum* type. Death occurred eleven days after the occurrence of convulsions. At *post mortem* examination of the lungs purulent bronchitis was found.

CASE XII.—Mrs. D., aged twenty-six years, was pregnant for the first time. Her urine contained "one-twelfth solid albumin" and on her discharge from hospital merely a trace. Her blood pressure was 120 millimetres of mercury and rose to 140 millimetres just before convulsions occurred. On her discharge the blood pressure was 110 millimetres. The patient was admitted for revealed accidental *ante partum* hæmorrhage. Her blood urea was 26 milligrammes per hundred cubic centimetres, the estimation being made during convulsions. She was delivered by instruments of an eight and a half months' still-born infant. "Veratrone" and morphine were used.

CASE XIII.—Mrs. B., aged thirty-two years, was pregnant for the fifth time. Her urine contained "one-sixteenth solid albumin." Her blood pressure was 180 millimetres of mercury her blood urea was 24 milligrammes per hundred cubic centimetres during convulsions. The patient died undelivered. She did not regain consciousness. The cause of death was probably cerebral hæmorrhage. "Veratrone" was used, but had no effect on either the pulse or the blood pressure. The temperature before death rose to 41·5° C. (107° F.).

CASE XIV.—Mrs. H., aged twenty-three years, was pregnant for the first time. Her urine contained "two-thirds solid albumin" and was clear on her discharge. Her blood pressure was 160 millimetres of mercury and on her discharge 110 millimetres. Her blood urea was 30 milligrammes before convulsions started. Labour was induced and the patient was delivered of an eight months' living infant. Convulsions occurred while the patient was in hospital undergoing dietetic and general treatment. She became affected by pelvic cellulitis.

CASES XV., XVI. and XVII. are included under the heading of chronic nephritis. The patients suffered from typical eclamptic convulsions.

A brief analysis of the foregoing fourteen cases shows the following points of interest:

1. The majority of patients are young and *primiparæ*.

2. There were two deaths. Statistically this fact is not very important because the number of cases (seventeen) is too small. But the cause of the deaths is of interest. One died from pulmonary lesion and the other very probably from a cerebral lesion. Pneumonia or œdema of the lungs is par-

ILLUSTRATIONS TO THE ARTICLE BY DR. R. B. WADE AND DR. KEITH INGLIS.

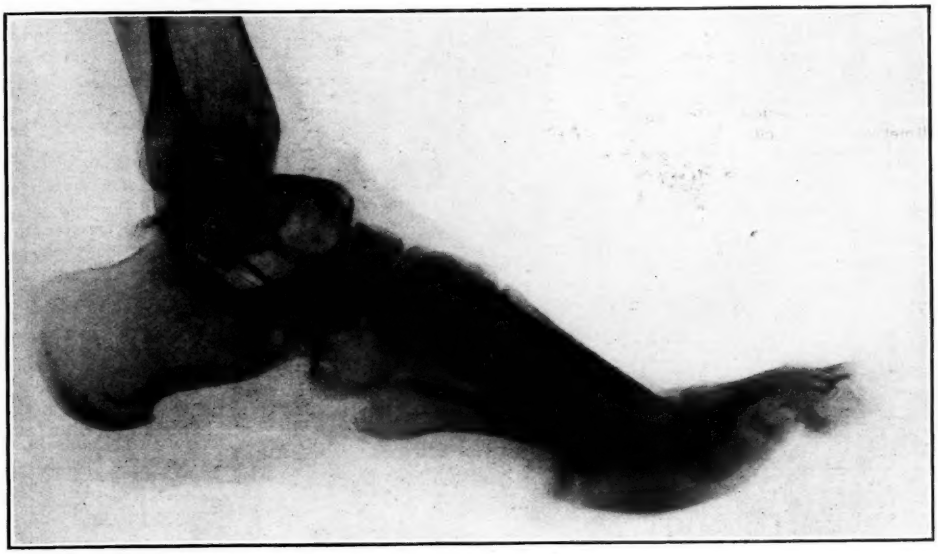

FIGURE I., Skiagram of Cyst of Astragalus.

FIGURE II., × 40. Wall of Cyst. Passing from the lumen of the cyst to the outer surface of the bone, that is from the left to right of the illustration, there is a dense fibrous lining then looser fibroblastic tissue with a few giant cells, then bone with fibroblastic tissue in the interstices. The outer surface of the bone is covered by connective tissue which here and there dips into the bone.

FIGURE III., × 40. One of the Small Fragments Projecting into the Lumen of the Cyst. It is composed of fibroblastic connective tissue in which are many cholesterin clefts.

ILLUSTRATION TO THE ARTICLE BY DR. ARCHIE ASPINALL AND DR. KEITH INGLIS.

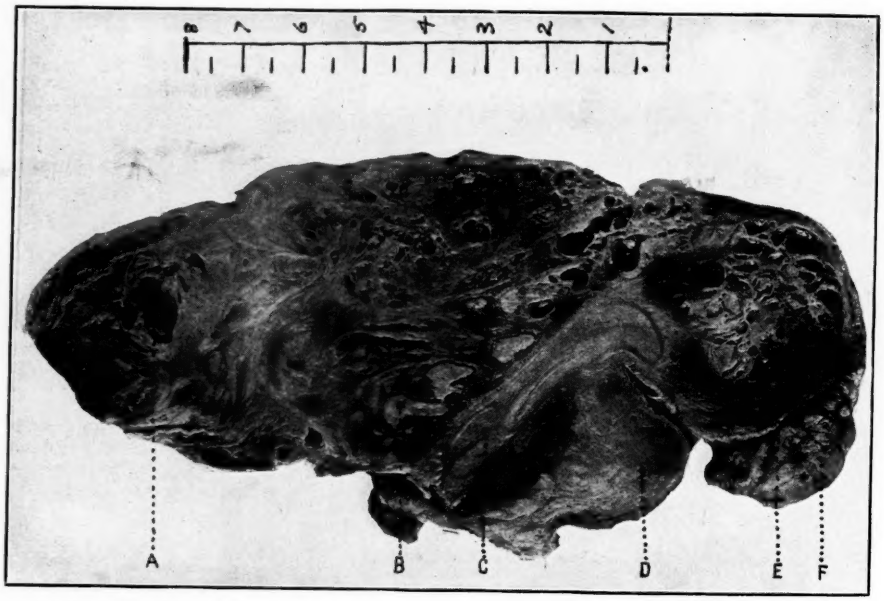

Figure Showing Sagittal Section Through Penis, Scrotum and Soft Tissues over the *Symphysis Pubis* (centimetre scale). A = Angiomatous subcutaneous tissue in the hypogastric region. B = Lymphangiomatous spaces containing purulent lymph coagulated by the fixative (Kaiserling's No. 1 Solution). C = *Corpus cavernosum*. D = Scrotum. E = Angiomatous tissue of prepuce as seen on section. F = Skin covering angiomatous tissue of prepuce.

ILLUSTRATIONS TO THE ARTICLE BY DR. E. H. STOKES AND DR. KEITH INGLIS.

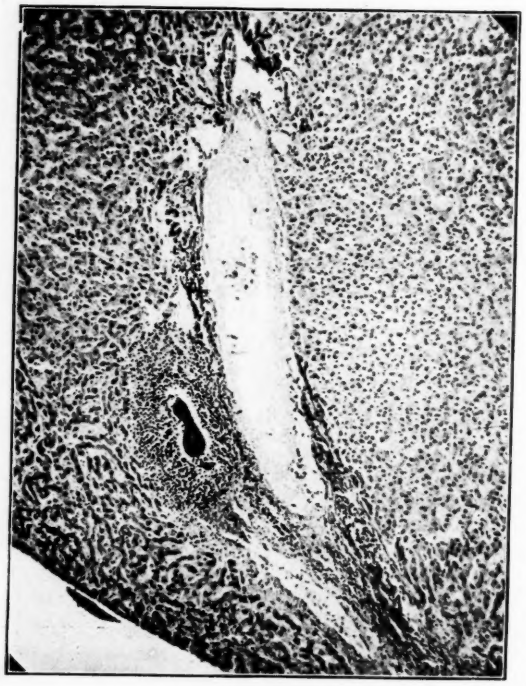

FIGURE I., × 70, Showing a Portal Tract. The large vessel is a branch of the portal vein. To the left of this vein in its upper part a small ill-defined bile duct may be seen. To the left of the vein near its lower end is an abscess with a mass of coagulated bacteria and *débris* in the centre. It is thought that this has been caused by bacteria in a branch of the hepatic artery.

FIGURE II., × 70, Showing Portion of the Glandular Mass Situated on the Upper Surface of the Liver. It is thought to be misplaced adrenal tissue.

ticularly severe in this type of case on account of the greatly exhausted condition of the patient. For this reason I think that the risks of ether as an anæsthetic for the minor manipulations are more serious than those of chloroform; but for a prolonged anæsthesia chloroform would be far more dangerous. Death from cerebral hæmorrhage, I should say, depends on the extent of the hæmorrhage. Severe hæmorrhage is preceded by a stage of cerebral irritation due to microscopical hæmorrhages. In those cases of cerebral irritation which are showing no improvement under conservative measures, I suggest as a method of treatment rapid delivery of the fœtus. This would mean Cæsarean section in most cases or if the cervix were fully dilated forceps delivery. I think that the delivery of the child and its attendant bleeding might possibly prevent the occurrence of a large cerebral hæmorrhage.

3. There were seven living infants delivered, that is 50%.

4. There were three cases of *post partum* eclampsia and in all three living infants were delivered.

5. The urine in the majority of cases was free of albumin on discharge of the patient from hospital.

6. The blood pressure fell to normal limits.

7. The blood urea estimation varied from twenty milligrammes to forty milligrammes per hundred cubic centimetres of blood.

Reports of Cases.

CYST OF THE ASTRAGALUS.

By R. B. WADE, M.D., Ch.M. (Sydney),
Honorary Surgeon, Royal Alexandra Hospital for Children; Honorary Surgeon, Royal South Sydney Hospital;

AND

KEITH INGLIS, M.D., Ch.M. (Sydney),
Director of the Department of Pathology, Sydney Hospital; Lecturer in Pathology, University of Sydney.

CLINICAL REPORT.
(R. B. WADE.)

MRS. H., (S.H. 5776), aged twenty-four years, gave the following history. In 1915 when fourteen years of age she felt acute pain in the right foot and the right ankle became swollen. The onset was sudden and occurred while she was sitting in a chair. When she arose from the chair, she limped and complained of severe pain in the sole of the right foot. She was sent to hospital for one month's "absolute rest," and was discharged from hospital improved. Soon the pain moved from the plantar aspect to the dorsum of the right foot over the head of the astragalus. The foot was often swollen, but there was no change in the colour of the skin.

In 1923 the patient knocked the anterior aspect of the ankle and was in bed for a week. From then on she was able to walk, but had pain after prolonged walking and, on sitting, the ankle became "stiff." She was unable to dorsiflex or extend the right foot fully and could not raise herself on the toes of the right foot; she had "no spring." Physical examination revealed slight shortening of the right leg, prominence of the head of the astragalus, swelling about both malleoli and pain on dorsi-flexion and plantar flexion.

X ray examination revealed marked cystic disease of the right astragalus, only a shell of bone remaining (see Figure I.). The other bones of the right foot were unaffected.

On April 28, 1925, astragalectomy was performed and the foot put up in backward displacement and very slight equinus. The result was functionally good.

A linear fracture was found along the inner border of the body of the bone.

PATHOLOGICAL REPORT.
(KEITH INGLIS.)

The specimens submitted for examination consisted of the astragalus in two pieces and the fluid from within the cyst.

The astragalus was merely a shell to the naked eye. No appreciable deviation from normal was evident on the outer surface of the bone, but on the inner aspect of the bony shell small soft fragments projected, giving this surface a slightly shaggy look, but in places the lining was comparatively smooth. Histological examination of the wall revealed the appearances seen in Figure II., which shows some relatively compact bone with fibroblastic connective tissue on either side as well as in the interstices within the substance of the bone. The lumen of the cyst is to the left of the figure.

The innermost zone of the cyst wall is composed of fairly dense fibrous tissue. Externally this merges insensibly in the fibroblastic zone which passes into the interstices of the adjacent bone. Some of the fibroblastic connective tissue has a loose almost myxomatous structure and in this part the vessels are dilated and have thin walls. Occasional giant cells are present, but they are very scanty.

The small opaque bodies projecting into the lumen of the cyst from the fibrous wall proved to be masses of cholesterin crystals situated in connective tissue which showed the usual giant cells and clefts. I think that probably these cholesterin deposits have no special significance, but correspond to the cholesterin crystals so commonly found in collections of fluid in other situations in the body, for example, in long standing hydrocœles of the *tunica vaginalis*.

On the outer surface of the bone there is a zone of connective tissue which in places is fibroblastic (see Figure II.), but elsewhere is densely fibrous. The vessels in this zone have thick walls. Processes of this connective tissue dip into the bone (see Figure II.).

The fluid from within the cyst did not clot on standing. The solid material settled at the bottom of the tube, leaving a relatively clear reddish brown supernatant fluid. Dr. H. S. H. Wardlaw found that the fluid contained methæmoglobin in solution. Unchanged pigment in corpuscles was also found. The amount of pigment present was too small to be consistent with the fluid being derived entirely from the blood. The protein content was 8%, a figure corresponding to the protein content of lymph or plasma. The fluid may be a mixture of blood and lymph.

HÆMANGIO-LYMPHANGIOMA OF THE ABDOMINAL WALL AND EXTERNAL GENITALS IN A CHILD.

By ARCHIE ASPINALL, M.B., Ch.M. (Sydney),
Honorary Surgeon, Sydney Hospital; Honorary Surgeon, Royal South Sydney Hospital;

AND

KEITH INGLIS, M.D., Ch.M. (Sydney),
Director of the Department of Pathology, Sydney Hospital; Lecturer in Pathology, University of Sydney.

CLINICAL REPORT.
(ARCHIE ASPINALL.)

J.J. (S.H. 2990), a male, five years of age, was admitted to the Sydney Hospital on January 24, 1923, complaining of a tumour in the inguinal region which had been present

since birth. A diagnosis of strangulated hernia had been made. The father stated that three months after birth the mass diminished in size and later it swelled up and subsided again and again. There were two swellings, one above the umbilicus and one in the inguinal region. The tumour never caused any inconvenience and did not increase in size. A week before admission to hospital the child fell down and hurt the tumour; since then there had been much pain and discomfort.

Physical examination revealed a large tumour extending over the lower abdomen and the *symphysis pubis* and involving both inguinal regions, the penis and the scrotum. The swelling was painful to the touch and of a bluish colour. The penis was large, red and œdematous. On the left side of the abdomen was a big bluish birth mark. A flattened swelling just to the left of the umbilicus felt like adipose tissue. A diagnosis of infected lymphangioma was made and foments applied.

The respiratory, circulatory and nervous systems manifested no abnormality. The temperature on the day of admission was 38·9° C. (102° F.). It varied between 37·8° C. (100° F.) and 39·4° C. (103° F.) until death.

On January 30, 1923, a small incision was made into an abscess in the inguinal region and pus was evacuated. Another incision was made into the tumour at the lower end of the scrotum and clear amber coloured fluid was obtained. Films made from the pus contained Gram-positive large and small bacilli, Gram-negative small bacilli and staphylococci. Cultures yielded Gram-negative bacilli (coliform) and a few staphylococci. From the cultures a vaccine was prepared and two doses were administered later. On February 2, 1923, as the temperature continued high (38·9° C. or 102° F.) many incisions were made into the tumour; from some of the incisions a clear yellow fluid escaped.

On February 27, 1923, the left side of the tumour was opened up and clear lymphatic fluid was obtained.

On March 2, 1923, the patient died.

PATHOLOGICAL REPORT.

(KEITH INGLIS.)

The *post mortem* examination was made in conjunction with Doctor Neil Goldsworthy. Only a partial examination was permitted by the relatives.

The body appeared wasted, the abdomen was disproportionately prominent especially on the left side. The penis and scrotum formed a big bluish-pink mass many times the normal size. There were several incisions in the tumour.

As far as could be judged from the examination made through the diaphragm the thorax was normal.

The lungs were a little pale and showed a moderate amount of œdema. There was a small hæmorrhage on the surface of the right lung.

The heart was of normal size. There was no pericarditis; the muscle was pale; the mitral valve was involved in a large red and white clot; when this clot was detached a small but definite degree of acute endocarditis was evident.

The liver appeared normal, perhaps a little enlarged.

The spleen was enlarged and soft as in acute infections.

The kidneys were pale and on section presented the appearance of cloudy swelling or catarrhal nephritis.

The alimentary tract, the pancreas, the pelvis and urinary bladder were all normal.

The brain and cord, thymus and suprarenal glands were not examined.

In regard to the tumour the left side of the abdominal wall was much thickened and on section showed a hæmangiomatous structure. Portions of the tumour resembled varicose veins; it was very superficial and did not extend beneath the deep fascia; no connexion with the large abdominal vessels could be made out; it spread from about the level of the left nipple above to the perineum below, and from the mid-line in front to the left mid-axillary line. The skin was discoloured brownish-pink above, but a bluish-pink below. In the upper part over the sixth to the ninth left ribs five to seven and a half centimetres (two or three inches) from the mid-line a collection of small pockets of pus was found in the tumour; in the lower part a clear serous fluid exuded from it.

Films were made from the pus and contained a few short chains of streptococci and some staphylococci; no tubercle bacilli were seen. The scrotum, penis and the soft tissues over the *symphysis pubis* were removed in one mass and a sagittal section made through it. The appearance of the cut surface is illustrated in the figure. The body of the penis can be seen passing upwards and to the right; below this is the scrotum not much affected; above and to the right is the very angiomatous prepuce; above and to the left is much angiomatous tissue which was situated over the *symphysis pubis* and lower part of the abdominal wall. Almost all of these cystic spaces contain lymph and not blood, but many of them are so infected that they contain almost pure pus which has become solidified by the fixative (Kaiserling's solution).

The association of hæmangioma with lymphangioma is interesting. I believe this to be common and consider that the vascularity found in the walls of cystic hygromata in the neck by Bland Sutton,[1] Thompson and Keiller[2] and others is an essential part of the lesion and not merely an incidental accompaniment. The tumour in this child was probably of the nature of a malformation; apparently it did not increase in size at a greater rate than the rest of the body; the way in which it alternately swelled and subsided is not unusual in vascular tumours. It is well recognized that cystic hygromata are susceptible to bacterial infection and this tumour seems to have been similarly prone to suppuration. In the cervical tumours the inflammatory reaction occasionally results in the obliteration of the cystic spaces and disappearance of the tumour; in this patient, however, the lesion was far too extensive to be satisfactorily dealt with in this way. The fact that non-infected cystic spaces intervened between those containing pus indicates that communication between some of the cysts, if present at all, was at least not free.

The sequence of events was probably as follows: The child was born with a malformation of both blood and lymph vascular systems. The lesion was susceptible to infection and the blow further contributed to make the tumour a *locus minoris resistentiæ* which was infected by organisms, gaining entrance either by way of the blood or from the body surface. The infection did not remain limited to the tumour, but spread to the blood stream, subsequently causing acute endocarditis of the mitral valve.

REFERENCES.

[1] John Bland Sutton: "Tumours, Innocent and Malignant," Seventh Edition, 1922, page 177.

[2] James E. Thompson and V. H. Keiller: "Lymphangioma of the Neck," *Annals of Surgery*, April, 1923, page 385.

ANEURYSM OF THE HEPATIC ARTERY ASSOCIATED WITH ACUTE ENDOCARDITIS.

By E. H. STOKES, M.B., Ch.M. (Sydney),

Honorary Assistant Physician, Sydney Hospital; Honorary Assistant Physician, Saint George District Hospital.

AND

KEITH INGLIS, M.D., Ch.M. (Sydney),

Director of the Department of Pathology, Sydney Hospital; Lecturer in Pathology, University of Sydney.

CLINICAL NOTES AND POST MORTEM REPORT.

(E. H. STOKES.)

Clinical History.

THE patient, T.R., a male, aged thirty-five years, a tiler by occupation, fell ill on November 24, 1924. His previous health had been good. He suffered from scarlet fever and measles in childhood and was attacked by influenza during the epidemic in 1919. In July, 1923, he was affected by

acute bronchitis. At various times he suffered from epigastric pain which was attributed to gastritis caused by overindulgence in alcohol.

He was married, had three healthy children and there was no reason to suspect that he had ever been infected by syphilis. He had not suffered from either rheumatism or any disease of the heart.

He was first seen during his present illness on November 25, 1924, when he complained of cough and pain in the left side. On physical examination his temperature was 37·8° C. (100° F.) and his pulse rate was 120 per minute. There was a small patch of fine crepitations in the region of the sixth and seventh intercostal spaces just in front of the anterior axillary line on the left side.

His condition was diagnosed as pleurisy and he was sent home to bed. With treatment he improved and on December 2, 1924, about one week after the onset of his illness, although his temperature was still above normal, being 38° C. (100·4° F.), his pulse rate was 80 per minute and his chest was free from crepitations.

On December 4, 1924, the pulse rate rose to 104 and the crepitations returned at the original site, the temperature still being 38° C.

As his sister-in-law who had been living in the same house, was suffering from pulmonary tuberculosis in an advanced form, he was sent to the Saint George District Hospital for observation. On admission there were in addition to the crepitations scattered rhonchi all over the chest. His urine, examined on December 8, was acid in reaction, 1030 in specific gravity; it contained a fair cloud of albumin and on microscopical examination a few pus cells, occasional red blood cells, several hyaline casts and some coliform bacilli were found.

On December 11, 1924, his temperature was 37·8° C. (100° F.) and a mitral presystolic murmur was found.

On December 14, 1924, an aortic to-and-fro murmur appeared and the mitral presystolic murmur was replaced by a systolic murmur at the same area.

On December 18, 1924, the patient collapsed after a large hæmatemesis. An aortic to-and-fro murmur with a mitral presystolic (Austin Flint) murmur was present, the apex beat being displaced slightly downwards and outwards. The temperature was now ranging from 38·9° C. to 40° C. (102° to 104° F.). He was tender in the epigastrium.

On December 19, 1924, he was slightly jaundiced and bile appeared in the urine. The sputum was examined microscopically and did not contain any tubercle bacilli, but Gram-positive cocci (chiefly pneumococci) and Gram-negative bacilli were the chief organisms present.

On December 24, 1924, he was very jaundiced and drowsy.

On December 29, 1924, his temperature was 39·4° C. (103° F.). Rhonchi and crepitations were noticed all over the chest. The liver was very much enlarged, the lower edge being palpable seven and a half centimetres (three inches) below the costal margin. The Van den Bergh test gave a "delayed reaction."

On January 5, 1925, his temperature was subnormal and he was passing urine and fæces involuntarily. He was deeply jaundiced. The aortic to-and-fro murmur was present and there was dulness at the pulmonary bases with absence of breath sounds. There was only slight œdema of the feet. He died on January 7, 1925.

The blood was examined by culture on two occasions; no growth was obtained.

The temperature chart for the first week after admission showed several rises to 37·8° C. (100° F.) and one rise to 38·3° C. (101° F.). For the second week it was definitely remittent, rising to 39·7° C. (103·6° F.) on one occasion. It was never below 37·8° C. On the twelfth day after admission (December 18, 1924) it rose to 40·6° C. (105·2° F.) and fell to normal after the hæmatemesis. For the next week it was slightly lower, but often reached 39·4° C. (103° F.). Towards the end the temperature was subnormal.

Post Mortem Notes.

A *post mortem* examination was performed. The following were the findings:

On external appearance there was considerable jaundice.

The Heart.

The pericardium was normal. The left ventricle was slightly dilated, the walls being somewhat thinner than normal.

The aortic valves were deformed and almost wholly destroyed by organized vegetations; the mitral valves showed some thickening of the cusps; no vegetations were present. There were a few vegetations on the cusps of the tricuspid valves. The coronary vessels appeared to be healthy. Gram-positive lanceolate diplococci (pneumococci) were isolated from the cardiac vegetations.

The Lungs.

The pleural sacs contained fluid. There was evidence of lobular emphysema with surrounding areas of collapse and congestion. There was no sign of consolidation.

The Abdomen.

A tumour, about 6·25 centimetres (two and a half inches) in diameter, was found attached to the inferior surface of the liver, lying medial to the gall bladder. It was covered with blood clot and on closer examination it proved to be an aneurysm of the hepatic artery.

A thin white nodular mass triangular in shape which resembled a suprarenal gland both in naked eye appearance and size, was found on the upper surface of the left lobe of the liver. It was situated just to the left of the falciform ligament midway between the anterior and posterior surfaces of the liver.

The liver was increased in size and although not weighed, probably was about 2,700 grammes in weight. On section it showed signs of chronic venous congestion.

The capsule of the kidneys stripped easily, the cut surface was bile stained and the cortex was swollen. There was no evidence of either abscesses or infarcts in the liver, spleen and kidneys.

On examination of the interior of the stomach several submucous hæmorrhages were noticed and the veins appeared to be dilated.

Comment.

It seems probable that the primary focus of infection in this case was in the lung and that the causal organism was the pneumococcus which was found in abundance in the sputum. The enlargement of the liver was apparently toxic as indicated by the delayed Van den Bergh reaction and the histological findings in the liver (see Dr. Inglis's report). With regard to the aneurysm of the hepatic artery it is difficult to determine whether it was present prior to the final illness or whether it arose as a complication during that illness.

Judging by the thickness of the aneurysmal wall it seems that the condition was long standing, possibly congenital in origin. This theory receives collateral support from the presence of suprarenal tissue in an abnormal position. Further, the absence of infarcts in the various organs may be taken as evidence against embolism being the cause of the aneurysm.

Acknowledgments.

I wish to thank Dr. K. T. Helms, Dr. A. W. Main and Dr. C. G. Bayliss, of the Saint George District Hospital, for assistance in connexion with the notes and *post mortem* examination.

PATHOLOGICAL REPORT.

(KEITH INGLIS.)

Histological Findings.

The specimens submitted for microscopical investigation consisited of: (i.) Heart, (ii.) glandular mass (? suprarenal) from the upper surface of the liver, (iii.) piece of kidney, (iv.) piece of liver, (v.) aneurysm with portion of hepatic artery attached.

The Heart.

The heart shows septic endocarditis with large vegetations on the valve cusps as stated in the report of the *post mortem* examination. It was not examined further.

The Liver.

The liver shows some venous congestion of moderate degree. The liver cells near the centres of the lobules contain a brownish pigment, situated almost entirely within the epithelial cells some of which are degenerate. In view of the venous congestion some of this pigment may be blood pigment, but I am inclined to think that much of it is bile pigment. The portal tracts do not show much deviation from normal. In particular there is no indication of obstruction to the bile ducts or of cholangitis; it is true that some of the larger bile ducts are not quite normal in that they show a moderate grade of catarrh. The hepatic branches of the portal vein are dilated. Only one small abscess was seen in the section; this was situated in or near a portal tract and as the bile duct and portal vein in this area show no suppuration, it seems probable that the suppuration is in relation to the hepatic artery. (The abscess is illustrated in Figure I.).

The cells in the suppurative focus are mainly polymorphonuclear (pus cells). The dark central mass shows no definite structure, probably it is composed of fibrin, bacteria and necrotic *débris*.

The Kidney.

The kidney shows definite cloudy swelling and catarrh of the convoluted tubules, a few accumulations of small cells with round nuclei resembling lymphocytes are to be seen, but no abscesses are present in the specimen examined.

Mass from the Liver.

The white mass found on the upper surface of the liver is composed of epithelial cells and is surrounded by a fibrous capsule. The arrangement of the epithelial cells suggests suprarenal rather than hepatic structure; indeed no definite evidence of hepatic structure is to be seen, for there are no indications of portal tracts, central veins or hepatic lobules. On the contrary the epithelial cells tend to be arranged in columns at right angles to the surface in a way resembling that seen in the *zona fasciculata* of the suprarenal cortex. Immediately beneath the fibrous capsule there appears to be a resemblance to the *zona glomerulosa* of the suprarenal cortex. The tissue resembling suprarenal cortex constitutes the greater part of the mass, but near the centre of the mass the arrangement alters, the tendency for the cells to be arranged in columns is no longer present and the structure is not unlike that seen in the medulla of the human suprarenal gland; it is possible, however, that the central portion corresponds to the *zona reticularis* of the suprarenal cortex. The microscopical appearance of this mass of glandular tissue is seen in Figure II.

Aneurysm of Hepatic Artery.

The specimen which at the *post mortem* examination was considered to be an aneurysm of the hepatic artery, was five centimetres in diameter. When I received it, the greater part of its contents was absent. One portion of the wall was composed of compressed liver substance, elsewhere the wall was fibrous and contained numerous vessels and spaces with smooth linings. It was impossible to determine the anatomical relations of these vessels because they seemed to racemose in irregular fashion. Sections of that portion of the wall of the aneurysm which blends with liver tissue, shows compression of the hepatic parenchyma and this is separated from the lumen of the aneurysm by fibrous tissue and coagulated fibrin. In the fibrous tissue are proliferated bile ducts which form winding tubes. There is no indication of involuntary muscle or elastic tissue in this situation. The vessel from which the aneurysm apparently developed, shows a large lumen with localized patches of thickened intima, myxomatous in places. The muscle coat of this vessel is very thick and looks abnormal. The nuclei of the muscle cells are widely separated and are narrow and wavy. Elastic tissue is situated not only between the thickened intima and media, but also in scattered fragments throughout the muscle.

An interesting point is that thickening of the intima is present not only in the large vessel from which the aneurysm apparently developed, but also in several other medium sized vessels enmeshed in the fibrous tissue surrounding the aneurysm. Similar thickening of the intima is conspicuous in a necrotic vessel situated apparently in the wall of the aneurysm and surrounded by amorphous necrotic looking material akin to that in the lumen of the aneurysm. The explanation of this is not quite clear. Close to one portion of the wall of the aneurysm are many conspicuous nerve trunks which are abnormally large. Some of the nerves are of looser structure than normal nerves and some of them appear almost myxomatous.

The microphotographs are from preparations stained by hæmatoxylin and eosin. Elastic tissue was studied in sections stained by Weigert's elastic tissue stain.

Accessory Suprarenal Glands.

The abnormal situation of the suprarenal tissue prompted a survey of the literature on accessory suprarenal glands.

Swale Vincent[1] states that the term "accessory adrenal in mammals has been used in different senses." We must carefully distinguish, he says, between: (i.) Bodies composed entirely of cortical substance "accessory cortical bodies"; (ii.) bodies made up exclusively of medullary substance "chromaphil bodies"; (iii.) true accessory adrenals composed of both cortex and medulla.

The accessory cortical bodies represent by far the larger number of structures which have passed under the name of accessory adrenals. They are said to show the three zones characteristic of the cortex of the adrenals of mammals, but no chromaphil cells are present; there is no "medulla." The smallest of these bodies are microscopical; others may reach a diameter of a centimetre or more. They are found in the neighbourhood of the chief adrenal, sometimes embedded in other organs, in the retroperitoneal space or in the genital region. True accessory adrenals, containing both cortex and medulla like the main gland, are said to occur in the neighbourhood of the abdominal sympathetic and in the region of the body where the cortical elements first arise.

G. W. Nicholson[2] states that suprarenal medulla is said to be absent except in the nodules in the solar plexus. In four of his specimens there was a localized patch of cirrhosis of the kidney at one or more points immediately beneath the accessory suprarenal. Minute retention cysts were present in them all. Eight of his specimens showed a very conspicuous, wide, thin-walled vein, not surrounded by scar tissue, in this situation. In a linear accessory suprarenal, of which he possesses serial sections, a vein extended longitudinally across the whole of its deep aspect at its centre, separating it from the kidney. These appearances suggest that the displacement was caused by an inflammatory focus or by the presence of an abnormal blood vessel between the suprarenal and the kidney. Nodules of suprarenal cortex have been described under Glisson's capsule on the lower surface of the right lobe of the liver, but Nicholson has been unsuccessful in finding them.

I suspect that in the cases of misplaced suprarenal tissue noted by Nicholson the fibrotic patch was not inflammatory or to be more precise was not infective, but was the result of a malformation, the dilated vein also being part of the malformation. In our patient I suggest that malformation may have been an underlying cause not only of the misplaced suprarenal tissue, but also of the aneurysm of the hepatic artery.

Though its situation was very unusual, the glandular mass on the upper surface of the liver in our patient was apparently an accessory suprarenal gland. To the naked eye it resembled a normal suprarenal and microscopically the picture was consistent with this interpreta-

tion, but in view of the fact that the tissue was not chromated, it is quite likely that the small amount of central tissue which at first was taken for medulla (chromaphil tissue) may be cortical and correspond to the *zona reticularis* of a normal gland. At the *post mortem* examination the kidneys were examined, but unfortunately no special examination of the suprarenal glands was made. However, the general examination revealed nothing grossly abnormal in these situations.

Arterial Aneurysms.

The literature on aneurysm of the hepatic artery is not accessible to me, but aneurysms of the splenic artery and of the renal artery have much in common with aneurysms of the hepatic artery so far as ætiology is concerned and five recorded cases of the former and one of the latter will now be considered in detail.

E. A. Baumgartner and W. S. Thomas[3] recorded the notes of a female patient, forty years old, who had an aneurysm of the splenic artery. She gave a history of indigestion all her life and three attacks of severe loss of blood (hæmatemesis and melæna) during the last year of her life. It seemed hard to explain such severe gastro-intestinal hæmorrhages when there was no ulceration. The authors attributed it to pressure by the tumour, for the *vasa brevia* were found considerably dilated. They say that forty cases of this condition have been published in the last fifty years; in six of them there was endocarditis and in five of the six embolism was given as the cause of the aneurysm. In twelve cases there was hæmorrhage into the gastro-intestinal tract. In eleven cases there were infarcts in the spleen. In six cases it is mentioned particularly that there was no luetic history, findings or reaction to the Wassermann test. In over half of the cases either gastro-intestinal or intraperitoneal hæmorrhages occurred. Several authors mentioned that no elastic tissue was found in the walls of the aneurysms and this was true of the author's case.

H. B. Willoughby Smith[4] recorded a fatal case of retroperitoneal hæmorrhage from an unusual source. The patient, a female, thirty-five years of age, was nine months pregnant. After a hard day's work ending up by moving a mangle,. there was an acute abdominal pain followed by death. *Post mortem* a large quantity of fluid blood was found in the peritoneal cavity. The mesenteric vessels were normal and the spleen was normal. The tissues behind the gastro-splenic ligament were considerably ecchymosed and torn up. As far as could be determined by dissection a branch of the splenic artery had given way just before it had reached the hilum. All the organs were normal except the heart which showed some recent endocarditis on the mitral valve. The author pertinently asks why did this particular artery give way and what share in the event did the endocarditis have? Was it (i.) a previous embolism and consequent weakening of the vessel wall with rupture during the strain of moving the mangle or (ii.) was the passage of the embolus the determining factor of the rupture by its impact in the narrow part of the vessel during the period of increased pressure? Against the first hypothesis is the fact that there was no infarct in the spleen. There was no atheroma in any of the vessels to account for the weakening.

James F. Goodhart[5] recorded a case of arterio-venous aneurysm of the splenic vessels with thrombosis of the mesenteric veins and localized acute colitis in a female, forty-seven years old, who had borne ten children. Eight or nine years before death she had a severe illness lasting fourteen days and characterized by severe pain in the abdomen, generalized abdominal tenderness and diarrhœa. The pains recurred occasionally and were unassociated with any indiscretions in diet. Eight weeks before death she had a violent pain in the lower part of the back and later still abdominal pain, diarrhœa, hæmatemesis and melæna. *Post mortem* there was a swelling in the parotid region, containing pus. About ten centimetres (four inches) beyond the ileo-caecal valve there commenced intense congestion of the colon extending along the bowel for twenty centimetres (eight inches) and very clearly defined from the pale intestine above and below. The branches of the superior mesenteric vein going from this area were found to be full of adherent thrombus. The splenic vein at the hilum of the spleen was dilated into a cavity the size of a hen's egg and filled with laminated clot. On dissection a valvular communication was found between the splenic artery and vein about five centimetres (two inches) from the origin of the latter. The whole length of the splenic vein was dilated and tortuous and partially filled with *ante mortem* thrombus and the dilated sac in like manner contained *ante mortem* clot. The splenic artery was of very full size. The spleen contained several white infarcts. The heart and lungs were healthy and there was but slight atheroma of the aorta. Goodhart offers no explanation as to the cause of the communication between the two vessels.

R. S. Taylor and John H. Teacher[6] reported two cases of aneurysm of the splenic artery. Their first patient was a boy, fourteen years old, who had had several attacks of acute rheumatism. Large shaggy masses of vegetations in process of breaking down were present on all three aortic cusps and recent vegetations were present on the mitral valve. There was some old standing thickening of the aortic and mitral valves, but no adhesion of the cusps. There was a small aneurysmal dilatation at the commencement of the aorta involving the orifice of the right coronary artery. The brain contained a small area of softening in the left frontal lobe and a very large hæmorrhage into the right cerebral hemisphere. The spleen was greatly enlarged with deep puckerings of the surface due to old infarctions. There was also a large recent hæmorrhagic infarction. In the spleen there were also one large mass and several smaller masses of thrombus, apparently enclosed in dilated vessels. The large mass was clearly an aneurysm and was related to the one remaining branch of the splenic artery, which remained at the hilus. This branch entered the spleen close to the large aneurysm, into which it passed directly. For about the last six millimetres (quarter of an inch) of its course just before it was lost in the large aneurysm, the vessel was slightly tortuous and showed a very prominent dilatation of its lumen. The large aneurysm extended along under the capsule for rather more than two and a half centimetres (an inch) and ended in the cicatrix of an old infarction.

There was further adjacent to the large mass a medium sized mass of thrombus which could be traced, gradually increasing in size, right through this half of the spleen. It occupied a large globular dilatation lying close beneath the capsule. Along its course it gave off several smaller branches which were made out to be greatly dilated branches of the splenic artery filled with soft red thrombus. It appeared to represent another, possibly the main branch of the splenic artery.

The authors say the ætiology of the aneurysm can be only a matter of conjecture. There was no evidence either in the spleen itself or in the walls of the cavities of any recent acute inflammatory condition, only a few polymorphonuclear leucocytes being seen. The most feasible explanation they consider is that their origin was inflammatory in nature. It is impossible to decide whether they originated by infected emboli of the larger arterial branches or by organisms circulating in the blood and lodging in the spleen, there giving rise to a periarterial inflammatory condition. The small aneurysmal pouch at the commencement of the aorta did not show much change in the wall. There was slight increase of fibrous tissue, but no sign of any active inflammation.

Their second patient, a female, forty-three years of age, complained of discomfort and later pain in the left hypochondriac region fourteen days before death. *Post mortem* the heart and aorta were normal. About one metre of the first part of the jejunum was gangrenous and there was thrombosis of the portal vein and its branches and also of the mesenteric vein. The main trunk of the cœliac axis and the hepatic artery were clear, but there was thrombosis of the splenic artery from immediately beyond its origin. The spleen showed a large anæmic infarct, occupying fully half of the organ. This was of fairly recent origin, but definitely an older lesion than that of the intestines. The spleen was adherent to a much prolonged left lobe of the liver.

The splenic artery followed an even more tortuous course to the spleen than normal and it was distended with thrombus and showed globular dilatations which made it look like a thrombosed varicose vein. The first of them, about 1·25 centimetres (half an inch) in diameter, was situated about seven and a half centimetres (three inches) from the origin of the vessel and there were three others of irregular sacculated form situated close to the hilus of the spleen.

Microscopical examination was made of sections including several undilated arterial twigs at the hilus of the spleen and of portion of two of the aneurysms. The undilated twigs appeared to have thickened walls with a certain amount of endarteritis and fibrous changes in the middle coat. The walls of the aneurysms were fairly thick and fibrous with a certain number of round cells principally replacing the middle coat. The internal elastic lamina was not recognizable and it would have been scarcely possible to tell that they were of arterial origin apart from the dissection. As to the course of events the authors say that only suggestions can be made. It seems probable that the aneurysm had long been in existence and from the history it seems probable that the occurrence of the large infarction corresponded with the onset of symptoms. This may have been due to embolism, in which case the embolus probably came from the thrombus in one of the aneurysms or it may have been due to direct occlusion of the arterial branch by thrombosis of an aneurysm; the thrombosis gradually spread back to the commencement of the splenic artery, where it was arrested. About the same time thrombosis occurred in the splenic vein which, however, extended into the main portal trunk, producing the thrombosis of the entire portal system and giving rise to infarction of the intestine.

The tortuous course followed by the splenic artery in the second patient of Taylor and Teacher is interesting, because elongation and tortuosity of arteries is a recognized congenital malformation. John Cairney[7] studied the frequency, causation and type of tortuosity found in the cervical portion of the internal carotid artery. As regards the causation he says various views have been advanced. Firstly, that the condition is due to arteriosclerosis and increased arterial pressure in old age or disease and secondly, that it is congenital. To test this latter supposition Cairney examined twenty fœtuses and found definite tortuosity in five instances of one or both internal carotid arteries.

A more striking example of arteries that were unnecessarily long is reported by Thomas Lindsay.[8] The patient was a female, seventy-seven years of age, whose brachial arteries appeared to be congenitally too long for the upper arms. In full extension they were quite visible and resembled in shape a bent bow. With the arms flexed the arteries closed up like a U spring. The depth of the loop was about 6·25 centimetres (two and one-half inches). Though no mention is made of increased girth, the photographs suggest that the vessels were much larger than usual.

R. P. Rowlands[9] found a spontaneous aneurysm of a branch of the renal artery in a female, forty-seven years of age, who suffered from symptomless hæmaturia. The kidney was removed at operation. Black streaks of altered blood and an infarct were seen in the renal substance and a ruptured saccular aneurysm, the size of a large pea, was found partly on the surface of one of the lower renal papillæ. It arose from one of the branches of the renal artery. Rowlands says that aneurysm of the renal artery is very rare. Some are due to trauma, others are spontaneous. Atheroma, syphilis, embolism or infection appear to have been the causes in the few recorded cases of spontaneous aneurysm of the renal artery, but Rowlands found no evidence of any of these in his case.

At this juncture I wish to draw attention to a remarkable case reported by A. J. H. M. Paulin[10] of aneurysm of the pulmonary artery with acute endocarditis engrafted on a congenital heart lesion.

The patient, a female child, had had extending over the whole of the right half of the face an immense nævus which had been treated by thermo-cautery. When six years and ten months old she died from acute endocarditis after an illness of three or four weeks' duration. Pneumococci were grown from the blood before death. *Post mortem* it was found that the pulmonary artery was replaced by an aneurysm extending from its origin to its division into two branches. The branches were thrombosed and the aneurysm was filled by organizing adherent blood clot. Both lungs contained infarcts. The pulmonary valves were the site of lesions typical of recent valvular endocarditis. There was a perforation in the middle of the upper part of the interventricular septum and narrowing of the infundibilum (*conus arteriosus*). Microscopical evidence suggested to Letulle who made the histological examination, that the dilatation of the vessel was chronic, but it was impossible for him to say what was the nature or age of the chronic lesion. The author concludes that the patient suffered from a dilatation situated above the narrowing of the pulmonary artery, the dilatation being transformed into an aneurysm by an ulcerative pneumococcal panarteritis.

Here then we have a patient with a congenital vascular lesion of the face, a congenital lesion of the heart and a dilatation of the pulmonary artery which was probably congenital. I am inclined to think that all of these lesions were due to faulty development and not to inflammation during intrauterine life. These malformed tissues are prone to inflammation and when blood infection supervenes, they are the areas chosen to bear the brunt of the attack. The sequence of events in this case is probably akin to that obtaining in the case of hæmangio-lymphangioma of the abdominal wall and external genitals recorded elsewhere in this issue.

Discussion.

The most striking feature in our case is the association of endocarditis with an aneurysm of the hepatic artery. In a critical review on bacterial endocarditis Irving Simons[11] states that embolic aneurysms are due to one of two factors: either a large fragment of vegetation from the heart valve is carried suddenly and lodges in an artery or there is an infection of the arterial wall (by way of the *vasa vasorum*) by bacteria of low virulence which are circulating in the blood. It is, he says, impossible to decide which is the method of pathogenesis; at any rate a thrombus is formed and finally an aneurysm results.

A Stengel and C. C. Wolferth[12] in a paper on mycotic (bacterial) aneurysms of intravascular origin state that bacterial infections may invade the vessel wall either from the outside or from within. Those coming from without are usually due to the extension of infection to the vessel walls from a neighbouring or contiguous inflammatory focus. In those coming from within the vascular apparatus embolism is frequently a factor. It is now generally believed, they say, that when an embolus plugs the lumen of an artery, inflammatory processes begin on the inner surface and spread outward. The formation of an aneurysm is dependent on partial disintegration of the wall, particularly the internal elastic lamina.

Infection, I suppose, must be regarded as the most likely explanation of the aneurysm in our patient. Some of the facts, however, seem to me to warrant a certain amount of hesitation in accepting this interpretation:

(i.) The autopsy revealed an abnormally situated glandular mass, probably adrenal, pointing to developmental peculiarity in this patient.

(ii.) In none of the organs were there infarcts and it seems remarkable that the only fragment of vegetation to get away should choose the hepatic artery. Weakening in the vessel wall due to infection carried by the *vasa vasorum* is of course possible, but there is no proof of this.

(iii.) Of the forty cases of aneurysm of the splenic artery collected by E. H. Baumgartner and W. S. Thomas[3] endocarditis was present in only six. In many of the cases there were splenic infarcts and no endocarditis. I suggest that these infarcts were caused by thrombosis due to disease of the splenic artery and were not caused by embolism.

(iv.) Certain features of these cases of aneurysm of the splenic artery are consistent with congenital malformation and are difficult to account for as the result of infection alone. These features are:

(*a*) In the second case of R. S. Taylor and John H. Teacher[6] the splenic artery followed an even more tortuous course to the spleen than normal and showed globular dilatations which made it look like a thrombosed varicose vein. The spleen was adherent to a much prolonged left lobe of the liver. There was no endocarditis and the aneurysms seemed to be of long standing.

(*b*) Elongation and tortuosity of arteries is a recognized congenital malformation.

(*c*) In a case of spontaneous aneurysm of the renal artery R. P. Rowlands found no evidence of atheroma, syphilis, embolism or infection.

(*d*) In the case of hæmangio-lymphangioma reported in this issue by Dr. Aspinall and myself, we have an example of a developmental abnormality part of which consisted of dilated vascular tubes like varicose veins. This lesion remained quiescent for five years and then following trauma became infected. The infection involved the blood stream and was associated with a small but appreciable degree of infective endocarditis of the mitral valve.

(*e*) Paulin's case of aneurysm of the pulmonary artery associated with acute endocarditis engrafted on a congenital heart lesion provides an even closer analogy.

It is possible that a similar sequence of events took place in the present example of aneurysm of the hepatic artery associated with endocarditis. This, however, is uncertain.

Summary.

(1) A case of aneurysm of the hepatic artery is reported.

(2) *Post mortem* examination revealed bacterial endocarditis of the aortic valve and a glandular mass regarded as accessory adrenal tissue on the upper surface of the left lobe of the liver.

(3) The question as to whether the bacterial endocarditis was primary and the aneurysm infective and secondary or whether the aneurysm was present prior to the onset of bacterial endocarditis is discussed.

References.

(1) Swale Vincent: "Internal Secretions and the Ductless Glands," Third Edition, 1924, pages 144 to 152.

(2) G. W. Nicholson: "Studies on Tumour Formation," *Guy's Hospital Reports*, July, 1922, pages 340 to 343.

(3) E. A. Baumgartner and W. S. Thomas: "Aneurysm of the Splenic Artery," *Surgery, Gynecology and Obstetrics*, October, 1924, page 462.

(4) H. B. Willoughby Smith: "A Fatal Case of Intraperitoneal Hæmorrhage from an Unusual Source," *The British Medical Journal*, January 14, 1911, page 83.

(5) James F. Goodhart: "Arterio-Venous Aneurysm of Splenic Vessels with Thrombosis of Mesenteric Veins and Localised Acute Colitis," *Transaction of the Pathological Society of London*, Volume XL., 1889, page 67.

(6) R. S. Taylor and John H. Teacher: "Two Cases of Aneurysm of the Splenic Artery," *Glasgow Medical Journal*, April, 1911.

(7) John Cairney: "Tortuosity of the Cervical Segment of the Internal Carotid Artery," *Journal of Anatomy*, October, 1924, page 87.

(8) Thomas Lindsay: "Elongation of Brachial Arteries," *The British Medical Journal*, November 22, 1924, page 669.

(9) R. P. Rowlands: "Aneurysm of a Branch of the Renal Artery: Rupture: Nephrectomy: Recovery," *The British Medical Journal*, November 22, 1924, page 939.

(10) A. J. H. M. Paulin: "*Étude d'un Cas d'Anévrisme de l'Artère Pulmonaire avec Endocardite Aigue chez une Enfant*" (*Greffés sur une Cardiopathie Congénitale*), *Vigot Frères*, Paris, 1922.

(11) Irving Simons: "Critical Review: Bacterial Endocarditis," *Quarterly Journal of Medicine*, April, 1914, page 291.

(12) A. Stengel and C. C. Wolferth: "Mycotic (Bacterial) Aneurysms of Intravascular Origin," *Archives of Internal Medicine*, April 15, 1923, page 527.

Reviews.

ANDREAS VESALIUS.

"THE Iconography of Andreas Vesalius," by M. H. Spielmann is the third of the research studies in medical history published under the auspices of the Wellcome Historical Medical Museum.[1] It was originally intended to form part of a *Liber memorialis* projected in 1914 and dedicated by the people of Brussels in honour of the four hundredth birthday of their great compatriot Vesalius.

Mr. Spielmann, whose similar study of the portraits of Shakespeare is so well known, has here undertaken a most careful and complete review of all alleged portraits of Vesalius whether engraved, painted or sculptured.

Anatomy in the fifteenth century had scarcely varied for over one thousand years, the teachings of Galen which were based on the dissection of dogs and other animals, still persisted as the absolute authority. Vesalius insisted on the prime importance of the direct investigation of the human body. Born in Brussels, 1514, Vesalius was a professor at twenty-three and when only twenty-eight years of age published his epoch making book, "*De Humani Corporis Fabrica*" which overthrew many Galenian superstitions and established anatomy on a modern basis.

By a strange coincidence the same year, 1543, saw another epoch making book published by Copernicus which demolished the ideas of Ptolemaic astronomy with its central world and concentric spheres on which ancient medical astrology and prognosis so often relied.

Both met with vigorous opposition among their peers, their own generation received them not; but within a century their methods had been accepted as the basis of these sciences as we know them today. England was one of the first countries to follow Vesalius, thanks to the energetic efforts of his contemporary, Dr. Caius (or Keys), who refounded Gonville and Caius College, Cambridge, in 1588, and was President of the Royal College of Physicians. A famous man in his day was Caius and well enough known to be the subject of a caricature by Shakespeare. For his own college he obtained the privilege of the right to dissect two bodies within its precincts every year.

Natural curiosity leads us to the desire to get some impression of the personality of so great a genius as Vesalius, a pathfinder in medicine, a pioneer in science and it is surprising to learn that out of about twenty-nine portraits in oil and forty-nine engravings, together with various medals and sculptures, alleged to represent Vesalius, the only truly authentic portrait is the brilliant woodcut from Calcar's drawing which adorns his great work on anatomy, the *Fabrica*. Vesalius is there shown to be a short, rather stockily built man, with a large round head and broad high forehead, presenting a prominent frontal curve. He wears a moustache and square beard and has thick curly hair. Dark, keen eyes and heavy eyebrows, a rather short nose, slightly uptilted at the end and full lips complete a countenance full of vigour and intelligence. Beneath the portrait is the motto: "*Ocyus jvcvnde et tuto.*" Altogether he is essentially Flemish in type. Spielmann claims that he belongs to the same round-headed Celtic type as Shakespeare and Keith is quoted as stating the association of this typical roundhead or brachycephalic type with certain mental qualities. Even in ancient Greece Myres tells us the dark haired roundhead Alpine type represented the artistic and cultural element of the land.

The artistic level of the portrait and the anatomical drawings are very high. Joseph Calcar, the artist, was a pupil of Titian and his collaboration with Vesalius was singularly successful.

Though Vesalius in later years was court physician to Charles the Fifth, no direct portrait in oils is accepted by the author. Several are accepted as oil studies after

[1] "The Iconography of Andreas Vesalius," by M. H. Spielmann, F.S.A.; 1925. London: John Bale, Sons & Danielsson, Limited. Crown 4to., pp. 265. Price: 30s. net.

the woodcut, but the majority on straight out physiognomy are obviously not portraits of Vesalius. The Bâle "portrait" for example has a Roman nose!

Others and here the author excels himself by his profound knowledge and experience in art and antiquarian research are excluded for various reasons. Either they were painted at a later date or the painter could never have met his supposed sitter or the previous history of the painting puts it out of court. For example, in one well-known "portrait" by Calcar in the Louvre the coat of arms is quite distinct from that of Vesalius who sported three weasels at speed—Vesalius is the latinized form of *Van Wesele*—thus showing the ascription to be untenable and proves the portrait to be that of a Melchior von Brauweiler.

Eleven alleged portraits are to be found in the United Kingdom—probably due to the influence of Dr. Caius—and of these eleven only two resemble the anatomist and neither of these were painted from the life, but are merely honest memorial pictures.

Of the twenty-nine oils twenty he says to all except optimists and zealots cannot command acceptance.

Apart from the authenticity of the portraits and their enumeration the book is full of delightful little excerpts on customs, medical and otherwise or concerning incidents of the life and times of Vesalius. Among them one may note in reference to a painting of Vesalius dissecting a living person in Spain that Spielmann describes it as an absurd legend, the falsity of which has been completely proved. From an artistic point of view and faithfulness to the original woodcut one is impressed by the stately monument at Brussels by Geefs, the bust by Godechaile and the engravings by Onghena and Steinla.

Mr. Spielmann's volume deserves a welcome from all lovers of the historical study of medicine and should be of the greatest service in eliminating these counterfeit portraits and preserving to us a true picture of the personality of a great genius of our craft.

EMERGENCY SURGERY.

EMERGENCY SURGERY is a term which to the Australian mind would not be sufficiently embracing to cover the full scope of the work by Dr. George de Tarnowsky.[1] In fact, it would be difficult to find just the term for the book. "Industrial Surgery" would possibly be better .

But it is more than that and covers a great deal of prophylactic, plastic and general surgery. Frank admission is made that much of the work has been built up and culled from labours during the recent world war. For example the work of Cushing and other men on head injuries is fully quoted and the well-known illustrations by that master artist A. K. Maxwell will be welcomed by those who know the merit of his work.

Blood transfusion and shock are both fully dealt with on modern lines, but the consideration of the nature of shock is not deeply entered into, the theories being merely mentioned. This perhaps is wise! The symptomatology and treatment are good.

In the treatment of cranial injuries, as already mentioned, Cushing's work is freely drawn upon and the grouping of signs and symptoms for diagnosis is to be approved and should be of great service. With its aid the diagnosis of the severity and location of cerebral injuries is made possible by those who have not the benefit of expert knowledge of neurology.

Plastic surgery may seem out of place in a book bearing such a title as the present volume. Nevertheless the subject of face, eyelid and jaw wounds is quite extensively and well expounded. The same may be said of injuries to nerves.

The chapter devoted to the consideration of abdominal wounds is excellent. We do not mean that there is advocated anything of a new or startling nature, but the advice is sound in both investigation and treatment.

The handling of the injuries to the spine is masterly and the author goes quite fully into the subject without undue waste of words. Unfortunately the treatment of the paralysed bladder is practically side stepped.

Fractures and dislocations are considered in detail and the subject is intelligently discussed. The chapter forms a very handy work of reference, but we would like the author to have told us what to do with recurring dislocation of the lower jaw.

The chapter on splints, like most of the work, is excellent.

The book, though published in America, is written in English, therefore its perusal does not jar.

The work is freely illustrated with really good plates and drawings and the paper is of excellent quality. The volume may be thoroughly recommended as being sound and exhaustive.

[1] "Emergency Surgery: The Military Surgery of the World War Adapted to Civil Life," by George de Tarnowsky, M.D., F.A.C.S., D.S.M.; 1926. Philadelphia: Lea and Febiger. Royal 8vo., pp. 734, with illustrations. Price: $7.50 net.

A TEXTBOOK ON PHYSIOLOGY.

IN the preface to the first edition of Bainbridge and Menzies' "Essentials of Physiology" it is stated by the authors that their object in writing the book was "to bring together in a concise form the fundamental facts and principles of physiology."

This is well maintained in the fifth edition of this book edited by Dr. C. Lovatt Evans.[1] It is somewhat larger than its predecessors, but the relative space allotted to each section is on the whole well judged and the subject matter dealt with in a satisfactory manner. Perhaps a more detailed treatment of the chapter dealing with the chemistry of the body would be advisable. A knowledge of biochemistry is so essential to the understanding of physiology that in a book of over five hundred pages more than two pages should be allotted to the discussion of the chemistry of carbohydrates and fats.

The additions to the book being mainly interstitial, it is impossible to discuss them all, but those under tissue respiration are instances of the many recent assets.

To those students who require a simple, but sound outline of physiology this book can be highly recommended.

OTO-RHINO-LARYNGOLOGY FOR NURSES.

WE note with satisfaction the appearance of the second edition of "Ear, Nose and Throat Nursing," by Dr. Hardie Neil.[2] The book is a handy and practical little volume and emanates from the sister Dominion of New Zealand. Designed primarily for the use of senior nurses it should prove quite as useful for the general practitioner. Operations and methods of treatment are simply and concisely described in addition to the special duties for the nurse. Indeed it is almost more a book for the doctor than for the nurse; as a book on which lectures might be based, it would be useful. There are several excellent diagrams and some useful photographs. Mention is made of Dr. R. H. Godsall's operation for frontal sinusitis. The reader is left with the impression of an emphatic and practical personality, a man who knows what he wants to say and how to say it. The book is clearly printed and can be recommended with confidence.

[1] "Bainbridge and Menzies' Essentials of Physiology"; Fifth Edition, Edited and Revised by C. Lovatt Evans, D.Sc. (London), M.R.C.S., L.R.C.P., F.R.S.; 1925. London: Longmans, Green and Company. Demy 8vo., pp. 508, with illustrations. Price 14s. net.

[2] "Ear, Nose and Throat Nursing," by James Hardie Neil, D.S.O., C. de G., M.B. (New Zealand), M.R.C.S. (England), F.A.C.S.; Second Edition; 1926. Auckland: Clark and Matheson, Limited. London: H. K. Lewis & Company, Limited. Post 8vo., pp. 106, with illustrations.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 11, 1926.

Passive Acquiescence.

IN his admirable book, "Medical Follies," Dr. Morris Fishbein devotes a chapter to some of the absurdities of the public health legislation in force in certain of the United States of America. He dares to hold up to ridicule the health authorities of these States and to castigate them for their inefficient administration and for their ignorance of the elementary principles of preventive medicine. Dr. Fishbein's action has been applauded because it has led the public to realize that the health of the community can be improved only if those responsible for the control of preventible disease are educated in hygiene and have initiative and imagination. Some of the public health laws in Australia are obscure, irrational and based on beliefs and superstitions. The measures adopted in the attempt to stem the spread of infective disease and to raise the general standard of health by the authorities in some of our States are almost as foolish and futile as the American methods to which Dr. Fishbein's caustic pen refers. The task of safeguarding the health and lives of human beings is one of the utmost difficulty and it is incomprehensible how the public can tolerate so important and complicated a service being made the tool of politicians.

The Royal Commission on Health in its excellent report deals with the prevention of disease and outlines a model scheme for the effective combating of the causes and spread of disease. In another part of the report some skeleton proposals are set forth concerning the cooperation of the practising portion of the medical profession and the health authority. Both the scheme and these proposals are based on the recommendations included in the Hone-Newland report. The Royal Commissioners agree with the Federal Committee of the British Medical Association in Australia that the unit of the medical side of the administration should be the general medical practitioner. In February of this year the Federal Committee referred the report to the Branches of the Association and at the same time asked the Federal Government to give it an opportunity of considering any proposals for legislation arising from the report before they are submitted to Parliament. A great deal of space of this journal has been devoted to a discussion of the report and in our issue of May 22 we made an appeal to the Branches to lose no further time in taking steps to ascertain what the mass of the profession is prepared to do to give effect to the recommendations of the Commissioners. The Federal Committee has quite recently had before it the replies of the Branches. In the main the Branches have approved of the report. A few critical remarks have been made; one or two amendments have been proposed. But there is not one word of constructive policy. The outline of the model scheme is left in its rough state, a basis on which a practical preventive medical service can be built. The medical profession has more than a passing interest in the evolution of this service. Hitherto the care of the health of the people in a collective sense has been regarded as being a function of the departmental health authority. The failure of the present system is largely due to the fact that the general practitioner is not an active integral part of the system. If the campaign against disease is to be properly organized and effectively launched, the general practitioner who stands in intimate relationship with the individual, must be enlisted in the army. Before the question of fresh legislation is considered, the details of the model scheme must be worked out and exact methods must be devised for carrying it into effect. The Commissioners have suggested some duties which should be imposed on general practitioners. These suggestions are of the nature of examples rather than a full list of duties. The task of evolving a complete scheme in its last details must be undertaken by someone. The nature of the service to be rendered by the general practitioner will have to be defined and the scheme will have to contain provisions for the coordination of the efforts exercised by the medical profession and for methods of control. There is ample scope for in-

genuity and for the gift of initiation. The Royal Commissioners have dealt with the general principles of modern hygiene; it remains for the medical profession or the politician to apply these principles. If the profession fails to produce something constructive, the terms and conditions of the service will be prescribed by the health authority and these terms and conditions may be less acceptable than those which the profession itself might have propounded. The most important points that need consideration, are the measures for discovering and attacking the sources of infection and for influencing the susceptibility of each section of the community to certain common diseases, the problem of the disabling effect of disease on the individual and the possibility of preventing an infection from passing from one person to another without interfering with the ability of the individual to earn a living. The duties of the general practitioner will not be occasional, subsidiary duties which he can perform at odd intervals between visits to private patients. They will form the most important of his professional actions. If the plan be well laid, the district health officer will be able to adjust the work in such a manner that it will not interfere greatly with the practitioner's private work. It is disappointing to find that the mass of the profession has evinced so little real interest in this subject that not one of the six Branches of the British Medical Association in Australia has prepared a complete programme of preventive medicine with a clear definition of the obligations of the general practitioner. It will be a national service, like the Army Medical Service in time of war. If the regulations are drawn up by non-professional persons, the service will probably be less acceptable to the medical profession and less efficient than if it is based on a programme devised by the profession.

Current Comment.

ECLAMPSIA.

In his admirable clinical study of the albuminuria of pregnancy, preeclamptic toxæmia and eclampsia, published in this issue, Dr. A. J. Cunningham has made a comprehensive survey. He gives good support to his contention that the three conditions should be regarded as different stages of the one disease. As Dr. Cunningham points out, this view had been advanced on previous occasions. In fact, the suggestion has been made that there may be some factor common to these three conditions and the lethal affection known as acute yellow atrophy of the liver. It is probable, however, that acute yellow atrophy of the liver is infective in origin and it is unlikely that any common factor of ætiological significance will be found.

Several points of importance emerge from Dr. Cunningham's study. In the first place the necessity for antenatal care is again emphasized. This is the more important in view of such statements as those of Kellogg on what he terms recurrent toxæmia of pregnancy. He holds that certain patients manifest symptoms of definite toxæmia and that it is impossible between the pregnancies to establish a diagnosis of chronic nephritis or renal insufficiency. He believes that 14% of women with toxæmia in one pregnancy who are not suffering from chronic nephritis, will manifest similar symptoms in another pregnancy. Kellogg expressed the conviction that the second appearance of symptoms could be prevented in half of the patients if adequate antenatal care were practised. It is clear that antenatal supervision cannot entirely prevent the occurrence of toxæmic symptoms and it is a matter of common experience that, although treatment applied early usually results in the recovery of the mother, this does not always occur and death of the fœtus is relatively frequent. The second point requiring emphasis is the paramount urgency of determining the ætiology of the threefold condition. Even if a given method of treatment were to result in almost 100% of cures, the necessity for determining the exact cause or set of causes would still be important. Too much attention is given at the present time to search for cures instead of to research into ultimate causes. This is the attitude of the lay mind and medical practitioners are apt to fall into the same method of thinking.

Dr. Cunningham enumerates many of the hypotheses which have been advanced in regard to the causation. Although he does not pin his faith to any one of these, he points out that the action of the poison resembles that of the poisonous albumins, proteoses, albumoses, peptones and so forth. He touches but lightly on the chemical changes produced in the blood and dwells more on the prognostic import of variations in the blood urea content. His study may well be considered in conjunction with a recent communication by Dr. H. J. Stander and Dr. A. H. Radelet.[1] They refer to their previous findings that the kation ratios in the blood of eclamptic women are within normal limits, with the possible exception of the phosphorus-calcium ratio which is slightly elevated. They suggested previously that this ratio might be associated with the hyperglycæmia sometimes found

[1] *Bulletin of the Johns Hopkins Hospital*, June, 1926.

in this disease. They have now studied the non-protein nitrogen, sugar, carbon dioxide combining power, uric acid, lactic acid and inorganic salts of phosphorus. They find that pregnancy is accompanied by a slight increase in the non-protein nitrogen and in the carbon dioxide combining power. This has previously been reported by others. They can discover no difference between the lactic acid values of non-pregnant and pregnant patients. They find that eclampsia is associated with certain definite changes. There is a pronounced tendency towards hyperglycæmia. The uric acid content and the lactic acid content are increased. In certain cases the carbon dioxide combining power was very low.

The number of patients observed was small. They comprised six normal non-pregnant women, seven normal pregnant women and six who suffered from eclampsia. It is not necessary to refer to the figures in detail, but the findings in one case are of sufficient interest to be mentioned. One patient who died twenty-three hours after admission, had been in coma after having had four fits at home; she had had no antenatal care. On account of the high level of the blood sugar and the relatively low carbon dioxide combining power twenty-five units of "Insulin" were given. This was followed by a decrease in the blood sugar, lactic acid and inorganic salts of phosphorus and by a slight rise in the carbon dioxide combining power. The high uric acid content of the blood was but slightly affected by the "Insulin" and it gradually rose to 12·2 milligrammes per hundred cubic centimetres just before death. "Insulin" likewise had no effect on the nitrogenous retention as evidenced by the high non-protein nitrogen. At *post mortem* examination the liver was found to be greatly damaged and Dr. Stander and Dr. Radelet express the opinion that at the time of the patient's admission too great liver damage had already been sustained. They regard it as probable that the "Insulin" prevented the development of an acidosis with great increases in blood sugar and lactic acid. It must be remembered that the administration of "Insulin" in this manner is more or less empirical. The hyperglycæmia was probably due to the liver damage. It is quite conceivable that "Insulin" may so tide a patient over a crisis by reducing a hyperglycæmia and preventing acidosis that the well known recuperative power of the liver is enabled to come into action. It was decided to test the suggestion that increase in the lactic acid content of the blood was solely due to the muscular activity incident to the convulsions. The blood of a patient suffering from epilepsy was tested a few minutes after a violent convulsion. The non-protein nitrogen, the sugar and the carbon dioxide combining power were within normal limits and the lactic acid figure was but slightly elevated. The lactic acid level was far higher in the eclamptic patients than in the epileptic. Dr. Stander and Dr. Radelet point out that the increase in lactic acid (200% or more above the normal level) takes place immediately after a convulsion and that in eclampsia there is no interference with the elimination of lactic acid. They suggest that a disturbance in the liver may be responsible for the increase in lactic acid and uric acid. There are three possibilities which may explain a high lactic acid content in the blood. It may be that an abnormally large amount of carbohydrate is metabolized; there may be an interference with the "lactic acid to glycogen" step in the carbohydrate chain and it is possible that lactic acid may be produced from sources other than carbohydrate, such as protein.

Dr. Stander and Dr. Radelet conclude that they are not in a position to determine how far the eclamptic blood picture is dependent upon the lesion observed in the liver. While it is quite possible that many of the changes in the blood are occasioned by the liver damage, it is necessary to go further back and discover what brings about the hepatic lesions. Until further investigations add to clinical and experimental data, it is probably justifiable to conclude that one of the amino acids or some other dissociation product of protein is primarily responsible.

THE USE OF "LIPIODOL" IN THE SPINAL THECA.

SINCE Sicard introduced his method of localization of spinal tumours by the injection of "Lipiodol" into the spinal theca, it has been used with success by many observers. It will be remembered that Dr. J. F. Mackeddie recently published in this journal an account of his experiences with this diagnostic agent. Most writers on the subject have agreed that the use of this drug in the spinal theca is free from dangerous results, at the same time little opportunity has been presented for studying the ultimate fate of the "Lipiodol." "Lipiodol" is an oily fluid consisting of *huile d'ouilette* containing about 40% of iodine. It is made from seeds of *Papaver somniferum* and no narcotic or alkaloid enters into its composition. There may be a danger that the diagnostic use of "Lipiodol" will meet with the fate that has attended so many new procedures, that it will be used without adequate indication and to such extent that it will be discredited. In these circumstances it may be well to draw attention to some recent investigations by Dr. Franklin G. Ebaugh and Dr. Hugo Mella.[1] They studied the effect of "Lipiodol" injections in thirteen persons. In all there was evidence of an "aseptic meningitis." This was indicated by a cellular increase in the spinal fluid, by an increase in its protein content and the frequent finding of blood in fluid drawn off by lumbar puncture. The changes were of a transient nature and the tendency was for recovery to occur within three days. It was found that recovery was delayed most in patients suffering from general paralysis of the insane.

[1] *The American Journal of the Medical Sciences*, July, 1926.

Abstracts from Current Medical Literature.

DERMATOLOGY.

Lupus Vulgaris Verrucosis.

F. C. COOMBES (*The Urologic and Cutaneous Review*, July, 1926) reports a case of *lupus vulgaris verrucosis* as a direct extension from a tuberculous hip joint. The writer points out that while lupus is not uncommon as an extension from tuberculous glands, the occurrence of the condition following bone infection is exceedingly rare. The case reported was in a female child, aged ten years, who had an abscess over the hip joint evacuated at one and a half years of age, three years and five years of age. The skin condition appeared at three years of age and at ten years was fairly extensive, covering the greater part of the buttock and spreading almost round to the vulva anteriorly. X ray examination revealed rarefaction about the acetabular cavity. The patient was given one skin dose of X rays every month for several months with definite improvement, but she then disappeared.

Fordyce's Condition of the Penis.

W. H. GOECKERMAN (*Archives of Dermatology and Syphilology*, July, 1926) states that while Fordyce's disease which was described in 1896 and which is not uncommon on the inner aspect of the lips, is of no importance in this region, it may become of considerable importance on the genitalia. The small, irregular, light yellow, closely aggregated, milium-like bodies are very rarely found on the mucous membrane apart from the oral cavity. In the case reported both mucous membrane and skin were affected. Histologically the circumscribed structures were made up of tissue typical of the sebaceous gland including its alveolar arrangement. However, a basal membrane and ducts appeared to be absent. In a comment on the case the writer calls attention to the fact that genetically Pringle's disease and Fordyce's condition are probably closely related.

Bromide Eruptions.

H. HALDWIN-DAVIS (*The Post-Graduate Medical Journal*, July, 1926) in a description of various forms of bromodermia gives examples of several very puzzling cases. He emphasizes the point that the salient characteristic of cutaneous bromodermia is the production of pus, the commonest example being bromide acne. In other forms the lesions are extensive and take on a somewhat warty appearance with pus exuding from a number of small openings. This is the "mycotic" or frambœsiform type. In infants the lesions are mostly nodular. In these cases the condition is generally passed on by epileptic nursing mothers. The author also describes the condition of congenital bromodermia; the eruption appears several days after birth and the author attributes this to the absence of staphylococci *in utero*. The most misleading forms are those with single lesions, three examples of which are related in detail. The first lesion was ulcerative; the second was warty with pus oozing from several points and adjacent to a small and scarred area. The third patient had arsenical pigmentation and keratoses as well as a small button-shaped bromide lesion on the inner aspect of the knee. This patient had been taking bromide and arsenic for twenty-three years, the latter probably being given to minimize the effects of the bromide. On discontinuance of the arsenic the keratosis diminished, but the other lesion became more angry in appearance. In conclusion the author draws attention to value of daily doses of sodium chloride in counteracting the effects of bromism.

Fox-Fordyce Syndrome.

H. GOODMAN (*Archives of Dermatology and Syphilology*, July, 1926) gives a short description of the condition described by Fox and Fordyce in 1902 and reports a further example. The two cases first described by Fox occurred in the axillary regions. The lesions were smooth, papular, numerous, firm and rounded. There was intense itching. Fordyce reported histologically (i.) hyperkeratosis of the orifices of sweat ducts and hair follicles; (ii.) dilatation of coil glands; (iii.) acanthosis; (iv.) chronic inflammatory changes. In the case reported the axillæ and *mons veneris* were affected. Pruritus in these regions commenced early after marriage. It is suggested that there is some relationship between this condition and the recently described apocrine glands.

Hereditary Hæmorrhagic Telangiectasis.

C. M. WILLIAMS (*Archives of Dermatology and Syphilology*, July, 1926) describes a classic case of hereditary telangiectasis. The patient's face was studded with dilated venules and spider nævi. In some areas large dusky red patches were noticed. There was a history of nose bleeding from childhood. Her father's eldest brother had a similar condition. The writer states that there are only thirty families reported in the literature as suffering from this condition and he lays stress on the fact that frequently other members of the family have been subject to hæmorrhages from the mucous membranes.

Irradiated Orange Juice.

H. L. MASLOW, D. H. SHELLING AND B. KRAMER (*Bulletin of the Johns Hopkins Hospital*, July, 1926) gives some tabulated results on the treatment of rhachitic rats with orange juice which has been subjected to the rays of ultraviolet light. The authors conclude: (i.) Antirhachitic properties can be imparted to orange juice by irradiation with the mercury vapour quartz lamp; (ii.) after feeding rhachitic rats with irradiated orange juice healing may be demonstrated as early as five days and is almost complete at the end of fifteen days.

Concurrent Herpes Zoster and Varicella.

A. C. ROXBURGH AND P. H. MARTIN (*British Journal of Dermatology and Syphilis*, July, 1926) report a case of *herpes zoster* and varicella in a man aged forty-one years. The herpes affected the second and third cervical nerves and was preceded by pain for three days. The rash affecting the trunk and limbs appeared two days later and appeared as a typical varicella eruption. The scabs of the herpetic lesions were the last to fall off. Three other similar case reports have been published recently, whilst cases have been frequently reported in which both conditions have occurred in dependants in different members of a family or in contacts. There is also serological evidence that both conditions have been caused by the same virus.

Association of Intestinal Indigestion with Various Dermatoses.

H. J. SCHWARTZ (*Archives of Dermatology and Syphilology*, May, 1926) publishes some statistics showing the frequency of the association of gastro-intestinal disturbances with dermatoses. The analysis of the fæces in nine hundred cases is given. Two hundred patients had been taking a Schmidt test diet and the remainder were given an average mixed diet; both groups gave parallel results. The most pronounced examples of protein putrefaction and carbohydrate fermentation occurred in patients who did not complain of any subjective symptoms. The results of examination of the fæces were recorded under the headings, macroscopical, microscopical and chemical findings. Special attention is drawn to the frequency of intestinal indigestion in severe acne, urticaria, *acne rosacea*, *eczema ani*, the oily type of seborrhœic eczema, furunculosis and pruritus. High figures were also obtained in *dermatitis herpetiformis*, *erythema multiforme*, pemphigus and neurodermatitis.

Schamberg's Disease.

L. K. MCCAFFERTY (*Archives of Dermatology and Syphilology*, July, 1926) reports a case of Schamberg's disease. According to the writer this is the second case reported in the United States and the fifteenth in the combined literature of England and the United States. It is interesting to note that all affected persons have been males and the condition is confined to the extremities. Some notes from Schamberg's description of his first case are given. The appearances are described as follow: "The patches are made up of pinpoint to pinhead sized reddish punctæ closely resembling grains of cayenne pepper, although perhaps of a slightly darker tint. They had somewhat of a telangiectatic appearance." *Angioma*

serpiginosum was mentioned by Whitfield as a possibility in a differential diagnosis. Kingery's histological report on his case was of great value. Its chief characteristic was a low grade inflammatory process with pigmentation in the upper part of the cornea. The pigment was found to be an iron derivative. In the writer's case the disease appeared about the ankles in a man, aged forty-four years. It had been present three years and was gradually spreading up the legs. Physical examination yielded no information. There is nothing in the literature referable to the treatment of this condition.

RADIOLOGY.

Cholecystography.

L. R. WHITAKER (*Journal of the American Medical Association*, January 23, 1926) gives his experience with cholecystography, including observations on the function of the gall bladder. The author uses the sodium tetraiodophenolphthalein salt and does not consider it toxic in the doses given. The intravenous dose is 0·04 gramme per kilogram of body weight and the drug is made into a 5% solution with distilled water, filtered and autoclaved and when ready for injection diluted to 2% strength with salt solution. Gravity apparatus is used and injection made very slowly. Great care is taken not to allow escape of solution into the subcutaneous tissues. No food is taken six hours before or ten hours after injection; at ten hours the part is examined by X rays and a skiagram is taken, the food is then given and a second photograph is taken and if the gall bladder is functioning, it should have contracted, thus furnishing evidence that it retains its elasticity. If no gall bladder shadow is obtained between ten and twenty hours, it is presumed that cholecystitis or cystic duct blockage is present. "Negative" shadows are obtained when gall stones are present. When orally administered, the pills are given in "enteric" coating and do not dissolve until they have passed out of the stomach. The dosage in these circumstances is 0·3 gramme per kilogram body weight. The pills are given after the evening meal and examinations are made about sixteen hours later. The oral method is almost, but not quite as reliable as the intravenous method.

Sella Turcica in Children.

M. B. GORDON AND A. L. LOOMIS BELL contribute a radiographic study of the *sella turcica* in sixty-four abnormal children (*Archives of Physical Therapy and X-Ray Radium*, April, 1926). The abnormal conditions met were Mongolian idiocy, myxœdema, thyreoid infantilism, hypothyreoidism, goître, spastic paraplegia and other gross deviations from the normal. One hundred and four normal children were examined as controls. The shape does not differ significantly in normal and abnormal children. Round and oval shapes predominate in children. There is no formation characteristic of any individual disease. The size of the sella has no relationship to the size of the head. Numerous tables with exact measurements illustrate this article.

Saturation Radiotherapy.

GEORGE E. PFAHLER (*British Journal of Radiology*, B.I.R. Section, February, 1926) describes his experiences with the "saturation" method of radiotherapy. The method consists of the delivery of an erythema dose into the diseased tissue, either as a single or a cumulative dose and then maintaining this effect for a certain time by means of additional smaller doses to correspond to the loss of effect during any given period. The author has given up the single massive dosage except in cases in which the disease is strictly localized and can be safely given an overdose. The saturation method is more difficult to administer than the single massive dose. It requires very careful measurement of the quantity of radiation, determination of both superficial and depth doses, accurate direction in cross-firing both in the primary and secondary doses and lastly the percentage value must be taken into consideration with each dose. A full erythema dose should be administered into the tumour within a week and this gives all the advantage of the massive dose and continues the effect during cell division. By this method less effect on the general organism is produced and better clinical results are obtained. Charts showing the percentage of radiations to be used at the individual sittings at different kilovoltages are given in the article.

Carcinoma of the Cervix.

R. E. FRICKE deals with the radiation therapy of carcinoma of the *cervix uteri* (*Archives of Physical Therapy and X-Ray Radium*, April, 1926). The technique is described. Careful examination, precise dosage, care in application and experience are necessary. Microscopical examination of sections should be made in all cases, a Wassermann test should be made and X ray examination undertaken to exclude metastases. Where cure is hoped for, massive dosage is recommended with repetition in eight weeks; in advanced conditions greater palliation is secured by fractional dosages. The patient is placed in the knee-chest position and the cervix well exposed by a Sims's speculum; radium emanation in one or two brass tubes (in rubber cots) is placed in the cervical canal and four to seven radium tubes in a gauze applicator are spread round the vaginal cervix. The rectum is protected by gauze-covered, six millimetres (quarter-inch) lead between the vaginal radium and the rectum. The dosage averages 3,000 millicurie hours for the first treatment. The parametria are treated by high voltage X rays through suprapubic, sacral and perineal ports. The author uses a water-cooled tube with forty milliampères of current at 240,000 volts, with one millimetre copper filter and one millimetre aluminium filter and fifty centimetre skin-target distance. A fairly severe reaction is sought.

Hepatic and Renal Function.

E. A. GRAHAM, W. H. COLE, G. H. COPHER AND S. MOORE contribute a joint paper on simultaneous cholecystography and tests of hepatic and renal function (*Journal of the American Medical Association*, February 13, 1926). The authors, on finding that the gall bladder could be defined after the ingestion of bromine and iodine salts of sodium phenolphthalein, attempted to determine the hepatic function by estimating colorimetrically the amounts of these salts retained in the blood after rendering the serum alkaline. The ordinary tetraiodophenolphthalein did not colour the blood serum sufficiently to be easily recognizable. The two isomeric iodine compounds behave differently in colouring the serum, the phenoltetraiodophthalein giving greater effect than the tetraiodophenolphthalein. Sodium phenoltetraiodophthalein is equally satisfactory for cholecystography. The best skiagrams are obtained four hours after intravenous injection of 0·055 gramme per kilogram of body weight. There is a greater tendency to local venous thrombosis with this salt. The amount left in the blood is estimated by the Rosenthal test and as the salt is also excreted by the kidneys, it is possible to estimate it colorimetrically in the urine after it has been rendered alkaline.

Cancer of the Œsophagus.

CHEVALIER JACKSON (*American Journal of Radiology*, December, 1925) pleads for earlier diagnosis of malignant conditions of the œsophagus. Œsophagoscopy and X ray examinations are the two methods of diagnosis. In the hands of the untaught œsophagoscopy is dangerous and misleading and it should be left to the expert. Examination by bougies is uncertain but with the œsophagoscope the lesion itself may be seen directly and the œsophagoscope should not be advanced unless it is seen that no pathological condition is ahead of it. If a pathological appearance is met with, a snipping may be taken for microscopical examination. The author prefers to make an X ray examination before applying œsophagoscopy. The act of deglutition is followed on the fluoroscopic screen and a very good estimate may be made as to the site of obstruction and its lateral extent. Stereoscopic skiagrams are often of great value. Absence of findings by any but experts in radiology is of no value. One important point which can be settled by fluoroscopy, is the question of whether aortic aneurysm is present or absent. The author describes several cases and illustrates the various appearances seen and describes various possible forms of palliative treatment.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Sydney Hospital on August 12, 1926. The meeting took the form of demonstrations by members of the honorary staff.

Fracture of Cervical Vertebræ.

DR. H. SKIPTON STACY showed a man, aged forty-seven years, a labourer, who had been hit in the back by a fall of stones whilst in a stooping position seventeen weeks previously. He had been stunned, but not rendered unconscious. His injuries had consisted of a hæmatoma over the left temporo-parietal region and minor abrasions. For several hours after the accident he had been able to move any of his limbs. When admitted to the ward two and a half hours later there had been complete paralysis of all four extremities, but no sensory involvement. Babinski's sign had been obtained on the left lower limb and no knee jerks had been elicited. At this stage the patient had been in a critical condition, respiration had been solely diaphragmatic in type. X ray examination had revealed a doubtful fracture of the fifth, sixth and seventh cervical vertebræ.

For the first fortnight the patient had suffered from retention of urine and this had necessitated the tying in of a catheter. Enemata had been given every second day to produce an action of the bowel. Two days after admission the patient's temperature had risen to 37·9° C. (100·2° F.) and three days later it had reached 38·9° C. (102° F.). The pulse rate had been 120 and the respiratory rate 32 in the minute. He had then become affected by hypostatic pneumonia and cystitis from which he recovered in two weeks. He had then made steady progress. Two weeks after admission the condition known as automatic bladder appeared. In three weeks the patient had begun to gain some power in his wasted limb. In four weeks he had begun to regain some control over his bladder and in six weeks control over both bladder and rectum had become complete. Motor power in the upper limbs had almost completely returned and was steadily improving in the lower limbs. At the time of the demonstration the patient was able to walk unassisted and looked the picture of health. A skiagram taken four and a half weeks previously revealed a fracture of the seventh cervical vertebræ. Dr. Stacy pointed out that the plantar reflex on the left side was one of dorsiflexion and on the right side plantar flexion. The knee jerk on the left side was described as +++ and on the right side +. No ankle or patellar clonus was detected. Some paresis of the right upper extremity was still present and the right hand shake was weaker than the left.

Neuralgia of the Dental Nerve.

Dr. Stacy also showed a married woman, aged fifty-five years, who had been admitted to hospital on June 5, 1926, complaining of severe trigeminal neuralgia on the right side. The pain had started twelve months previously when in the course of chewing she had suddenly been seized with a sharp shooting pain originating from the stump of the second right premolar and the first molar teeth. She had had numerous similar experiences and subsequently both stumps had been extracted, but with no improvement in her pain. The pain had become so severe and constant that she was afraid to eat. During the warm summer months the pain had left her, but with the onset of winter it started with renewed vigour. It had not been confined to the distribution of the right inferior dental nerve, the pain shooting forward along the lower jaw as far as the middle line. Later it had spread up the cheek as well. The patient had eight children, all strong and healthy; she had suffered from five miscarriages.

On examination the patient had appeared well nourished and healthy looking, but wore a haggard expression. She had pointed to the region of the right lower alveolar margin, previously occupied by the second premolar and first molar teeth as being the site of origin of her pain. There had been no teeth posterior to the incisors on this side. The region had not looked abnormal in any way and had not been tender to touch. No reaction had occurred to the Wassermann test.

On June 15, 1926, the patient had been submitted to operation under rectal anæsthesia. The gum covering the right alveolar margin posterior to the incisor teeth had been removed. The sharp edge of bone had been nibbled away and the mucous membrane oversewn with catgut. The specimen had been sent to the Pathology Department and a report had been received to the effect that macroscopically the subepithelial connective tissue seemed more bulky than normal and somewhat hyaline in appearance. On microscopical examination the connective tissue had appeared increased beneath the surface epithelium and in this fibrous tissue were many cells which appeared to be inflammatory. The nerves appeared more conspicuous than normal, but they were not as numerous as in a stumped neuroma. The tissue was definitely pathological, but the pathologist was of the opinion that the connective tissue increase was inflammatory rather than neoplastic in nature. The patient had made an uninterrupted recovery and had not experienced any pain since. She was profuse in her thanks for being relieved from a pain which, according to her, words could not describe.

Lead Poisoning.

DR. H. C. ADAMS showed a male patient, aged forty-four years, a labourer, who was suffering from lead poisoning. The patient had been born in Bathurst. Ten years previously he had begun to work in a bulk store, handling white lead, paints and brassware. Five years later he had begun to complain of a continuous dull pain in the region of the umbilicus. This had continued for one week and the patient had noticed that his wrists were weak. After obtaining medical treatment his wrists had improved and had remained comparatively well for six to seven months. Since then they had become progressively worse. He had suffered from occasional recurrences of the abdominal pain. The wrists had not been painful or tender to pressure. He had been removed from the paint department of his place of business to the wrapping department, where he had handled brassware and lead traps for four and a half years. On admission to hospital one month before the meeting he had complained of slight breathlessness, nocturnal frequency of micturition and constipation.

Dr. Adams pointed out that the patient had short, irregular nails due to his habit of biting them. He was a heavy drinker and smoker. There was extensive sepsis about his gums and a continuous blue discoloration at the junction of the gums and teeth. The abdomen was soft but tender. The pulse was regular, its tension was increased and the vessel wall was palpable. The systolic pressure was 146 and the diastolic pressure 110 millimetres of mercury. The cardiac system was normal. The pupils were equal and reacted to light and accommodation. The cranial nerves were intact. The abdominal reflexes were present. The knee jerks were increased. The plantar reflex was flexor in type. No ankle clonus was present. The patient had almost complete paralysis of the extensor muscles of both wrists. Slight paresis of the flexor muscles was present. The left wrist was involved more than the right. Sensation was intact. No motor or sensory changes were present in the lower limbs. The respiratory system was normal. Examination of the urine revealed the presence of lead. A very heavy cloud of albumin, present on the patient's admission, was gradually decreasing. No casts had been found in the urine and no red cells, but a few pus cells and motile organisms had been seen. The urea concentration test had been carried out. Before the administration of urea the percentage of urea had been 1·5%. One hour after administration of urea it had been 1·4%. Two hours after giving urea it had been 1·6% and one hour later 1·7%.

The patient's blood had been submitted to extensive investigation. A completely positive result had been obtained on submission of the blood to the Wassermann test. No reaction had been obtained from the complement fixation test for gonorrhœa. The blood urea was seventy-five milligrammes per hundred cubic centimetres of blood

and the blood creatinine was two milligrammes. A blood count taken one week after admission had yielded the following result:

Erythrocytes, 4,230,000 per cubic millimetre;
Hæmoglobin value, 50%;
Colour index, 0·6;
Leucocytes, 26,400 per cubic millimetre;
Neutrophile cells, 82%;
Lymphocytes, 15%;
Large mononuclear transitional cells, 3%.

The red cells had been normal in size and shape. Numerous examples of punctate basophilia had been seen. No nucleated forms had been found.

Two weeks later the blood had been examined again with the following result.

Erythrocytes, 4,370,000 per cubic millimetre;
Hæmoglobin value, 60%;
Colour index, 0·7;
Leucocytes, 15,500 per cubic millimetre;
Neutrophile cells, 65%;
Lymphocytes, 29%;
Eosinophile cells, 1%;
Transitional cells, 5%.

The red cells had manifested a good deal of achromia. No other abnormalities had been found.

The ocular fundi had been examined and no abnormality had been found.

Hemiplegia.

DR. G. C. WILLCOCKS, O.B.E., M.C., showed a girl, aged eight years, who was suffering from hemiplegia. This case will be reported in a subsequent issue.

Dry Skin.

Dr. Willcocks also showed a male patient, aged forty years, who had a very dry skin. The facial hair was scanty and the face rather dry and glossy. The patient stated that the dry skin had been in evidence since childhood. As his appearance suggested a subthyreoid state the basal metabolism had been estimated. This had been reported as + 33, which was very definitely above normal.

Dr. Willcocks explained that the patient was shown to indicate the apparent fallacy of regarding a clinically dry skin as evidence of a subthyreoid condition alone. He had recently had a patient whose appearance suggested a subthyreoid state and in whom the basal metabolism was — 30%. Treatment with thyreoid extract had made him feel worse and caused him to lose 6·3 kilograms (one stone) in weight which he could ill afford to lose. It was not on Dr. Willcocks's advice that the thyreoid administration had been carried out.

Chronic Pancreatitis.

Dr. Willcocks's third patient was a married man, aged forty-seven years. He was a car driver and had three children. He had complained of severe epigastric pain, four to five hours after food for twelve months. It had been relieved by alkalis and it was severe enough to double him up and prevent work. He had lost 15·7 kilograms (two and a half stone) in weight during the last seven months.

He had been operated upon in December, 1925, and the appendix had been removed, the gall bladder had been palpated and found healthy. The pain had not been relieved and the patient had continued to lose weight. There was some frequency of the bowel action.

The patient was flabby and obviously below weight. X ray examination had revealed no gastric or duodenal lesion. The urine contained sugar in considerable amount. The blood sugar was above normal. With starvation and a basal metabolic diet the glycosuria had ceased and the patient had gained weight steadily, alkalis had relieved the pain.

A fractional test meal yielded a high acid curve suggestive of duodenal ulceration. The serum had not reacted to the Wassermann test and examination of the stools revealed no definite abnormality.

In discussing the diagnosis Dr. Willcocks said that at first pancreatic neoplasm had been suspected, since X rays showed no evidence of duodenal ulcer. The gain in weight on a basal diet (3·1 kilograms or seven pounds gain in two weeks) was rather against this diagnosis. The patient manifested severe epigastric pain and glycosuria.

Some authors alluded to hunger pain in *diabetes mellitus*, but Dr. Willcocks was loath to ascribe his pain to diabetes alone without further observation. It was probable that ulceration of the duodenum existed not infrequently and was not evident on X ray examination. He had seen several such cases. In this patient there might be duodenal ulceration and diabetes, but it was more probable that some chronic pancreatic lesion existed causing the pain and glycosuria.

Fibrous Nodules (Arthritis Deformans).

Dr. Willcocks's last patient was a labourer, aged forty-one years, who complained of pains in the joints of the hands and wrists. He denied heavy potus and said that he had never had an attack in the big toes. There was deformity of the wrist joints and a fluctuant swelling over the lower radio-carpal joint, the finger joints appeared thickened and there were soft and hard nodules scattered over the fingers. The joint pains were probably due to *arthritis deformans*. X ray examination revealed no bony lesions. The nodules were probably fibromata. Trench stated that they were not uncommon in association with *arthritis deformans*. Osler held that they contained round and spindle cells. Treatment (local and general) seemed to have no effect on the pains or the nodules. Excision had not been practised, since there was no likelihood that the patient would be benefited. Dr. Willcocks had a similar patient in whom the nodules had existed for eight years.

Lupus Vulgaris Non Exedens.

A patient suffering from *lupus vulgaris non exedens* was shown conjointly by DR. LANGLOH JOHNSTON and DR. GEORGE R. HAMILTON. The condition (dry variety of *lupus vulgaris*) had been present for fifty-one years in a female patient, aged sixty-one years. It was explained that the dry variety of *lupus vulgaris* was that generally seen in Australia, due no doubt to the influence of the bright sunlight, whereas in England and Sweden especially the ulcerating variety was the variety generally seen. The lesions affected practically the whole of the right cheek and nose, the left cheek being involved to a small extent. The right upper lid and forehead were involved, this distribution being exceptional, as these parts generally escaped in *lupus vulgaris*.

The disease had started when the patient was ten years of age as a small red spot on her right cheek and had gradually extended till the whole of the right side of the face, nose, right upper and lower lids, forehead and left cheek were affected with a slightly infiltrated yellowish-red rash which was slightly elevated in parts.

On pressing out the blood from the affected parts with a glass tongue depressor or diascope the non-vascular apple jelly like spots of Hutchinson stood out clearly. The disease was spreading by a nodular margin. The early onset of the disease (ten years of age) and the presence of the apple jelly nodules distinguished the condition from that of *lupus erythematosus*.

Lupus Erythematosus.

Dr. Johnston and Dr. Hamilton also showed five patients suffering from *lupus erythematosus*; three were females and two were males. In all five patients the disease had appeared between the ages of twenty and forty. In one instance there was a doubtful history of tuberculosis. The disease affected the flush area of the face and ears and was bilaterally symmetrical; one patient (female) manifested the typical bat-wing distribution, the scalp also being affected. The affected regions showed an erythematous scaly eruption spreading by a slightly raised margin and in the centre atrophic scarring.

Progressive Hemiatrophy with Scleroderma.

Dr. Hamilton also showed a patient suffering from progressive hemiatrophy with scleroderma. An account of this case will be published in a subsequent issue.

X Ray Dermatitis.

Dr. Hamilton showed a patient who was suffering from X ray dermatitis following radiography. An account of this case will be published in a subsequent issue.

Ulcer of the Stomach.

DR. HOWARD BULLOCK showed a female patient six years after she had undergone an operation for ulcer of the stomach. According to the history the patient had suffered with pain after food for many years. This pain had commenced about ten minutes after the ingestion of food. It had lasted for half an hour to many hours and had been relieved by the patient lying on her back. On one occasion the patient had vomited blood, since then she had had no vomiting; there had been no melæna. The appetite was good, the action of the bowels regular, but the patient had lost a lot of weight. On examination the patient had appeared thin and pale; tenderness had been present in the middle line above the umbilicus.

On June 8, 1920, a bismuth meal had been given by Dr. J. G. Edwards and this had revealed an irritable stomach with hyperperistalsis and a six hours' residue. There had been tenderness on pressure over the *pars cardiaca* with spasmodic incisura due to reflex pylorospasm. a diagnosis of ulcer of the stomach had been made.

On June 17, 1920, operation had been undertaken under ether anæsthesia. Dr. Bullock said that he had excised a large ulcer involving the lesser curvature and the anterior and posterior walls of the stomach with its centre at the *incisura cardiaca*. A short looped posterior gastro-enterostomy had been performed towards the proximal end of the posterior wall of the cardiac portion of the stomach. In the performance of this operation two layers of plain catgut had been used internally and silk had been used for the suture of the sero-muscular layer externally. The pyloric canal had been closed by a ligature of kangaroo tendon. Appendicectomy had also been performed. In 1924 total hysterectomy had been performed on account of fibroids. The specimen removed at operation had been submitted to the late Sydney Jamieson for pathological examination and a report of "simple ulceration" had been received.

Dr. Bullock said that the following were points of interest in the case: (i.) The band round the pyloric canal had caused permanent occlusion of the pylorus. (ii.) The artificial stoma had been placed proximal to the seat of ulcer in case of future contraction and quite a large portion of stomach had been left distal to the stoma with no untoward result. (iii.) The patient had gained over 12·6 kilograms (two stone) in weight, she enjoyed perfect health and could eat any kind of food without the slightest discomfort. (iv.) The silk used had caused no trouble.

Ulcer of Stomach and Duodenum.

Dr. Bullock's second patient had been submitted to operation for ulcer of the stomach and duodenum ten months previously. He was a man fifty-nine years of age and had complained of pain with food for five years. According to the history taken on October 10, 1925, the pain had occurred from one-quarter to one-half hour after the taking of food. Vomiting had been frequent and had relieved the pain. Hæmatemesis had occurred on one occasion only; there had been no melæna. His appetite was good, but he had been afraid to eat and had lost much weight. On examination the patient had appeared very emaciated and general tenderness had been present in the upper part of the abdomen. On October 4, 1925, an opaque meal had been carried out by Dr. Cutler and Dr. Dight and this had revealed "chronic ulceration of the cardiac and pyloric portions of the stomach suggestive of chronic ulcer."

On October 12, 1925, operation had been undertaken at Sydney Hospital under ether anæsthesia. The distal half of the cardiac portion of the stomach, the antrum and pylorus and the first part of the duodenum had been removed and the cut end of the stomach had been closed. A long looped anterior gastro-enterostomy had been performed and jejuno-jejunostomy had been carried out between the afferent and efferent loops. The bowel had been sutured in three layers, the two inner layers with plain catgut and the sero-muscular layer with silk. Dr. Keith Inglis had examined the specimen removed at operation and had reported "simple ulceration."

Dr. Bullock said that it was of interest to note that three ulcers had been found at operation, one in the cardiac portion of the stomach, one in the antrum and one in the first part of the duodenum. The patient had gained 18·9 kilograms (three stone) in weight and could eat any kind of food.

Carcinoma of the Rectum.

Dr. Bullock also showed a patient seven years after operation for carcinoma of the rectum. The patient was a man, aged seventy-three years of age. On November 3, 1919, he had complained of the passage of blood with and between motions for about six months. The stools had been normal in size. Gradual loss of weight had occurred and the patient complained of a feeling of weakness. On examination the patient had appeared very pale and a mass had been palpable in the rectum on rectal examination. Operation had been performed under ether anæsthesia at Sydney Hospital on November 6, 1919. The rectum had been removed by the abdomino-perineal route after the method of Ernest Miles. Two distinct growths of the bowel had been found about 21·5 centimetres (nine inches) apart. The specimen had been examined pathologically by Dr. P. E. Walton Smith and he had reported it to be adeno-carcinoma. Dr. Bullock in discussing the case pointed out that the patient enjoyed excellent health and carried on his occupation of shifting and lifting hundred weight cases of white lead in a city house. He had full control of the artificial anus. No deep X ray therapy had been used and apparent cure had resulted from surgery alone.

Carcinoma of the Tongue.

Dr. Bullock's next patient had been operated on for carcinoma of the tongue three years previously. He was sixty years of age and when seen by Dr. Bullock in November, 1923, had complained of a sore on the tongue which had been present for eight months. The sore was getting larger and was causing pain. No reaction had been obtained on application of the Wassermann test. Treatment by potassium iodide had been of no avail. On examination a fungating mass had been present on the left side of the dorsum of the tongue. On November 7, 1923, operation had been undertaken under ether anæsthesia at Sydney Hospital. The submaxillary glands and upper deep cervical glands of both sides, the sternomastoid muscle and underlying cervical glands of the left side of the neck had been removed and then the left half of the tongue had been taken away. Pathological examination of the specimen by the late Sydney Jamieson had revealed the presence of squamous epithelioma. Dr. Bullock pointed out that after almost three years there was no sign of recurrence. He emphasized the fact that no deep X ray therapy had been used.

In contrast with the last mentioned patient Dr. Bullock showed a patient two years after operation for carcinoma of the tongue. The patient was sixty-one years of age. He had been seen by Dr. Bullock on October 26, 1924, and had complained of a growth on the tongue. This had been present for several months. The patient had previously been advised to avoid surgery and had been sent to another State for deep X ray therapy. X rays had been applied by an expert who was an advocate of this method of treatment. The result had been severe headache, prostration and increase in the size of the growth. On examination a huge fungating carcinoma of the dorsum of the tongue had been present and it had extended to the junction of the anterior with the posterior two-thirds of the tongue. Operation had been performed at Sydney Hospital under ether anæsthesia on October 30, 1924. The submaxillary glands, the upper and lower deep cervical glands and the sternomastoid muscles of both sides of the neck had been removed and then three-fourths of the tongue had been taken away. Dr. Keith Inglis had reported the growth to be squamous epithelioma. Dr. Bullock pointed out that deep X ray therapy had had no influence on the growth;

in fact under the influence of this form of treatment it had increased in size. There were no signs of recurrence.

Malignant Disease of the Mandible, Face and Chin.

Dr. Bullock's next patient was a man, thirty-two years of age. He had been seen for the first time in the out-patient department on December 20, 1925, complaining of a sore in the mouth which had been growing gradually for the previous ten months. A fungating mass had involved the floor of the mouth, the anterior part of the tongue and the alveolus and it had ulcerated through the left cheek. A report of squamous epithelioma had been received after examination of a snipping by Dr. Keith Inglis. The condition had been considered almost inoperable and the patient had been sent for deep X ray therapy. On June 25, 1926, a letter had been received from the deep X ray therapeutist to the effect that the growth was progressing after six months' treatment. On June 29, 1926, the mass had been larged and the mandible was necrosing, but the mass had been defined from the cervical tissues. Operation had been undertaken at Sydney Hospital under chloroform anæsthesia induced through a tracheotomy opening on July 8, 1926. Both external carotid arteries had been ligatured. The upper deep cervical glands had been removed and all the lower half of the face, chin and the skin of the neck to the hyoid bone, including the anterior three-fourths of the tongue and tongue muscles and the whole of the mandible. The remains of the tongue had been stitched to the surface. The growth had been examined by Dr. Keith Inglis and a report of squamous epithelioma had again been received. Dr. Bullock pointed out that deep X ray therapy had undoubtedly made operation possible. It had limited the spread and had been the means of clearing up the inflammatory swelling in the neck. The operation was mutilating, but the patient had wanted to live and his wife had also been anxious that the operation should be performed.

Carcinoma of the Stomach.

Dr. Bullock"s last patient was a man, fifty years of age, who had been operated on for carcinoma of the stomach four years previously. Seen on June 28, 1922, he had given a history of being quite well until December 24, 1921. On this day he had vomited after taking an iced drink. The next day he had passed a large quantity of blood and had continued ot do so for about eight days. When this ceased, he had commenced to suffer from slight fullness after meals. He had begun to lose appetite and weight. In May, 1922, five months after the onset, he had been admitted into one of the metropolitan hospitals for general examination and immediately after admission had passed pure blood for four days. An opaque meal had been given and sigmoidoscopy had been undertaken, but no abnormality had been discovered. Dr. Bullock said that the patient had been known to him personally for many years. He had seen at once that the man was obviously ill. An opaque meal had been given and this had revealed hyperperistalsis and pylorospasm.

On July 3, 1922, under ether anæsthesia fully three-fourths of the stomach had been removed on account of a huge growth of the lesser curvature involving the pyloric antrum and cardiac portions of the stomach. A short looped posterior gastro-enterostomy had been performed with the same technique as described in the previously mentioned cases. The late Sydney Jamieson had examined the growth for Dr. Bullock and the Department of Microbiology at the Board of Health had made an examination for Dr. A. A. Palmer. In each case the finding had been adeno-carcinoma. Dr. Bullock drew attention to several points of interest in the case. In the first place his personal knowledge of the patient was of considerable help to him in determining that the patient was really ill. In the second place there was freedom from all pain suggestive of previous ulceration. In the third place the X rays had not revealed a growth and the onset with bleeding was unusual. Finally the freedom from pain for four years was noteworthy.

Arterial Aneurysm.

DR. ARCHIE ASPINALL showed a male patient who had suffered from several aneurysms of the arteries in the lower extremities. An account of this case will be published in a subsequent issue.

Deformity of the Hands.

Dr. Aspinall also showed a patient whose hands were deformed as a result of an accident in childhood. This patient's condition will be recorded in a subsequent issue.

Hydatid Disease of the Innominate Bone.

DR. GEORGE BELL, O.B.E., showed a male patient, aged forty-seven years, who had been admitted to hospital on April 26, 1926. The patient had noticed a swelling of the left hip for six years. This had increased in size and had been neither painful nor tender. A year previously the patient became conscious of pain in the left limb below the level of the tumour. He had described this pain as being constant and of an aching character. At the time of admission the pain had extended to the left knee and had been shooting in character. Examination had revealed a cystic tumour extending from immediately above the left iliac crest to the great trochanter. The tumour had been smooth and not tender. A hard, loose body, about 2·5 cubic centimetres (one inch) in diameter had been palpable in the lower end of the tumour. This loose body had been freely movable. A skiagram taken on May 4, 1926, had revealed "cystic bone disease of the left ilium." On May 5, 1926, the complement deviation test, the precipitin test for hydatid and the Casoni test had been carried out, but no reaction had been obtained. The patient's serum had been subjected on two occasions to the Wassermann test and no reaction had occurred. The patient had then been seen in consultation with other members of the honorary staff, both physicians and surgeons. The consensus of opinion had been that the condition was one of hydatid disease of the ilium. Some, however, had favoured the diagnosis of "slowly growing cystic sarcoma" or a new growth of bone of low grade malignancy. It had been considered very unlikely that the condition was one of fibrocystic disease of the bone. Operation had been suggested as a means of arriving at a diagnosis. Dr. Bell said that he had hesitated to do this on account of the possibility of the subsequent infection of the lesion. On May 5, 1926, a general anæsthetic had been given and an attempt had been made to aspirate the contents of the tumour with a trocar and cannula. This had been unsatisfactory and a similar incision had been made over the upper portion of the tumour and a large quantity of fluid had been evacuated. The fluid had contained numerous small hydatid cysts and minute fragments of bone packed with minute cysts. The wound had been closed and had remained healed until May 19, 1926, when some fluid had escaped. Since that time fluid had continued to drain at intervals. It often contained minute hydatid cysts. The patient had been discharged at his own request on July 16, 1926. There was still some discharge from two sinuses in the wound.

On May 20, 1926, Dr. Keith Inglis had examined a portion of bone removed from the tumour and had reported that spaces between bony trabeculæ contained pink stained laminated material which was apparently hydatid ecto-cyst. Dr. Bell also showed skiagrams of the condition.

Thrombosis and Œdema Following Fracture.

Dr. Bell also showed a male patient, aged fifty-seven years, a labourer, who had been admitted to hospital on April 2, 1925. On that day he had been run over by a water cart which passed over both his ankles. A skiagram had revealed a fracture of both bones of the left leg in the lower third with slight displacement of fragments of the fibula. Swelling had been present with tenderness and some ecchymosis in the region of the fracture. Progress had been uneventful until April 17, 1925, fifteen days after the accident, when the whole of the left limb became greatly swollen and œdematous. No abnormality had been discovered in the urine and the circulatory system had been normal, except for the thrombosis. No reaction had recurred to the Wassermann test. The patient had not lived in the tropics. On May 11, 1925, the right foot had become œdematous and the following day Dr. Holmes à Court had seen the patient and had considered that

the condition was due to thrombosis. Dr. Bell said that he had seen thrombosis of the femoral vein following a fracture of the patella and on two occasions after excision of a prepatellar bursa. He had also been credibly informed of a case in which thrombosis of the left femoral vein followed an operation for fracture of the right patella. At the time of demonstration the patient had considerable œdema of the left leg with œdema of a less degree in the right leg.

Gun Shot Wound of the Abdomen.

Dr. Bell's third patient was a man about fifty years of age, a shooting gallery attendant, who had been admitted to Sydney Hospital on March 5, 1926. The patient had been shot in the lower part of the abdomen on the left side by an 0·22 rifle bullet in the shooting gallery. On examination a small punctured wound about six millimetres in diameter had been found just above the anterior superior iliac spine on the left side. Very slight muscular rigidity had been present in the region of the wound. The patient had been conscious and neither pallor nor cyanosis was present. The reflexes had been normal, the pulse rate eighty-six in the minute and the temperature 36° C. (97·8° F.); the respiration had been thoracic in character. Immediate operation had been undertaken by Dr. Bell. A mid-line incision had been made and a bullet was found embedded in the wall of the small intestine. The wall had not been quite perforated. The bullet was extracted and the wound closed with catgut. Double perforation had been found in the jejunum and this had been closed in a similar manner. Another part of the small intestine had been grazed. A fæcal odour had been noticed and a perforation found in the transverse colon. The wound had been closed with catgut sutures. The wound of entry had been found in the left side of the abdomen in the neighbourhood of the level of the brim of the pelvis. The peritoneum had been incised at this point and the left ureter found to be severed. The distal portion had been ligatured. The proximal part had been brought through a stab wound in the left loin and a catheter sutured into the ureter had been stitched to the wound. Dr. Bell had thought that recovery might be expedited if the left kidney were allowed to function temporarily.

The patient's condition had not permitted nephrectomy. The patient had subsequently made good progress and urine had drained freely from the tube. On April 7, 1926, the patient had complained of pain in the left shoulder and a friction rub had become audible over the lower ribs on the left side. Two days later the patient's temperature had been 37·8° C. (100° F.) and the base of the left lung was dull. Fifteen cubic centimetres of slightly turbid and very fœtid fluid had been withdrawn from the pleural cavity. Streptococci had been found in the fluid. On April 18, 1926, the patient had coughed up a large amount of very foul sputum. He had been discharged on May 12, 1926. On June 8, 1926, no cavity had been found on X ray examination of the lung, though the patient coughed up a large amount of sputum in the mornings.

Pyelograms and Urological Diagnosis.

DR. REGINALD BRIDGE showed a number of pyelograms illustrating a variety of lesions of the urinary tract. He also showed a number of recent pathological specimens corresponding to some of the pyelograms shown. Amongst the pyelograms were examples of early and late hydronephrosis, ureteral strictures and kinks, diverticula of the bladder, stones in the kidney, ureter and bladder and new growths of the kidney. Dr. Bridge pointed out that the pyelograms demonstrated the necessity of urological methods in diagnosis. He said that clinical signs were often very uncertain and quite absent and moreover many of the symptoms were common to other diverse lesions. Two specimens illustrated this in particular. The first was an infected hydronephrotic kidney of fifteen years' duration. The patient had dated the onset of symptoms from a pregnancy fifteen years previously. Ever since that time she had suffered from repeated attacks of cystitis. Bladder irrigations had been of no avail because the condition promptly recurred. Only on rare occasions had the patient complained of slight pain and discomfort in the left kidney region. Physical examination had disclosed nothing except pus in the urine and repeated examinations of the urine had failed to reveal the presence of tubercle bacilli. On cystoscopic examination extensive and severe cystitis had been found. The urine from the right ureter had been clear. The left ureter had been swollen and inflamed and there had been a purulent efflux. Pyelography had revealed an extensive dilatation of the whole ureter and a hydronephrotic kidney. Merely a shell of kidney tissue remained. The urine from this kidney had infected the bladder. The kidney and ureter had been removed and the bladder irrigated with "Mercurochrome."

The second specimen was a tuberculous right kidney from a man, thirty years of age. For twelve months he had suffered from severe bladder irritation. There had at no time been any symptoms referable to the kidney and physical signs had been absent. When first seen by Dr. Bridge the patient had passed purulent urine and had suffered from great frequency of micturition (every fifteen minutes). Tubercle bacilli had been found in the urine and a guinea pig injected with the urinary deposit had become infected by tubercle bacilli. Dr. Bridge showed the tubercles in the peritoneal cavity of the guinea pig. Cystoscopic examination had revealed clear urine from the left ureter and purulent urine from the right. The bladder had been inflamed, but no tuberculous infection had been evident, except some thickening of the right ureteral orifice. A kidney had been removed. Dr. Bridge showed the specimen and said that it was an excellent example of early unilateral renal tuberculosis.

Urological Instruments.

Dr. Bridge also showed an improved pattern of Bransford Lewis's tablet depositor for anæsthetizing the posterior portion of the male urethra. This instrument had an improved shape and size designed to take small tablets of "Borocaine" or other anæsthetic. He also showed an improved pattern of bladder retractor carrying a light. Both these instruments had been made for Dr. Bridge by Elliott Brothers.

Fractures.

DR. C. E. CORLETTE showed a series of lantern views of splints applied to various forms of fracture. He explained the indications for the use of each and described their method of application. He also gave a demonstration in the fracture ward.

Hæmochromatosis.

DR. E. H. STOKES showed a male patient, aged thirty-seven years, a blacksmith by occupation. In October, 1925, the patient had suffered from severe "cramps" in the legs which were worse at night; the pain used to shoot from the thighs to the toes. In November he had noticed that he was excessively thirsty and very hungry. He also passed large quantities of urine. Sugar, acetone and diacetic acid had been discovered in his urine and in January, 1926, he was admitted to hospital, becoming semi-comatose shortly after admission. He had improved under treatment by means of diet and "Insulin" and was discharged early in March. It had been noticed before discharge that his liver was considerably enlarged and that his skin was a slatey grey colour.

He had been readmitted to hospital early in April, 1926, and again treated by means of diet and "Insulin." His legs and scrotum had become œdematous. Several carious teeth had been removed and the œdema lessened. The urine had yielded the Prussian blue reaction, showing the presence of ferric salts. His blood sugar level had been considerably raised (the lowest figure recorded for the fasting blood sugar was one hundred and thirty-six milligrammes per hundred cubic centimetres). The glucose tolerance test had shown that he was suffering from severe diabetes. It had also been noticed that his renal threshold for sugar was considerably raised, his blood sugar often being over two hundred milligrammes per hundred cubic centimetres without the appearance of sugar in the urine. The largest quantity of urine passed in twenty-four hours had been three and a half litres (one hundred and twenty ounces). The microscopical

examination of his urine had disclosed a few epithelial cells but no blood, no pus cells nor casts were found. He had been discharged from hospital in May and had regained his normal weight.

The patient had been reexamined early in August, 1926. It had been found that his skin was still slatey grey; the lower border of his liver was palpable just above his right iliac crest; the splenic dulness was increased; the œdema of the legs was still present; tenderness was present in the calf muscles; his knee jerks were sluggish; his systolic blood pressure reading was 136 millimetres of mercury and the diastolic 90; the blood sugar (fasting) was 495 milligrammes per hundred cubic centimetres; the blood urea nitrogen was 16 milligrammes per hundred cubic centimetres; the blood creatinin 1·5 milligrammes per hundred cubic centimetres. The urea concentration test had given a normal result. At the time of demonstration the patient did not feel ill and was taking a diet which yielded 3,000 calories *per diem*; he was using twenty-five units of "Insulin" daily.

Meningo-Vascular Syphilis.

Dr. Stokes's second patient was a man, aged fifty-eight years. He complained that numbness in the legs had been present for two years; three months previously he had lost the use of his right foot. He suffered from giddy turns and occasionally would fall backwards. He had had no previous illnesses; he denied venereal disease. His wife had had six children and four miscarriages. He had been a seafaring man for a number of years.

On examination it was noticed that there was foot drop on the right side, evidently due to weakness of the *tibialis anticus* muscle. His right knee jerk had been present on the first examination three weeks prior to the meeting, but could not be elicited at the time of the meeting. His left knee jerk was absent. Both Achilles reflexes were absent and all sensations (light touch, pain, thermal, vibratory) below both knees were diminished. Rombergism was present. His pupillary reactions were normal. His *fundi oculorum* were normal. The electrical reactions of the leg muscles were normal.

A provisional diagnosis of meningo-vascular syphilis had been made and the usual pathological investigations to confirm the diagnosis undertaken. His serum yielded a positive response to the Wassermann test, as did his cerebro-spinal fluid. The cell count of his cerebro-spinal fluid was thirty-seven per cubic millimetre; the globulin reaction was present. The colloidal gold test (Lange) yielded a paretic type of curve.

Antispecific treatment had been inaugurated just prior to the meeting and the patient manifested a definite improvement.

A Case for Diagnosis.

Dr. Stokes also showed a male patient, sixty years of age, whose condition presented difficulty in diagnosis. On June 23, 1926, he had fallen a distance of one hundred and twenty centimetres (four feet) on to a steel deck, striking the antero-lateral aspect of his left shoulder on the deck. He had been taken to hospital and according to his statement, there was considerable bruising of his shoulder region. His arm had been bound to his side for a week, but when the bandages were removed, he found that he was unable to move the arm.

On examination of the patient it was noticed that there was wasting of the left deltoid muscle; he was unable to abduct his left arm. External rotation and adduction were also impaired. Flexion of the forearm upon the arm was weaker on the left side than on the right. There was slight impairment of sensation to light touch over the lower portion of the left deltoid region. His triceps and supinator reflexes were absent on the left side. The left deltoid muscle showed the reaction of degeneration. His left shoulder was examined by X rays soon after the accident, but no osseous lesion had been detected. A skiagram taken shortly before the meeting was reported upon by Dr. Edwards as manifesting ossification in the neighbourhood of the muscle insertions of the greater tuberosity of the humerus.

With regard to his previous history, it had been discovered that forty years previously he had suffered from a hard chancre, which had been treated for some weeks in San Francisco (the patient had been a seaman).

The blood serum gave a positive response to the Wassermann test.

On examining his nervous system in detail, it was found that the motor functions, with the exception of those mentioned above, were normal. Thermal sensation was delayed below the knees. Vibration sense was diminished in the legs. When his sense of passive position was tested, slight delay was noticed in the toes. His reflexes were normal except in the left arm; his pupils were equal and reacted normally; his knee jerks were feebly active; his Achilles reflexes were present. The *fundi oculorum* were normal. His cerebro-spinal fluid had been examined. A positive response to the Wassermann test had been elicited; the colloidal gold figures were of the luetic type; the cell count was fourteen per cubic millimetre, all the cells being lymphocytes.

With regard to the other systems, a trace of sugar had been discovered in the urine. The glucose tolerance test demonstrated that he was suffering from diabetes. Otherwise, no abnormality was discovered.

Dr. Stokes said that it was difficult to decide whether the disability was wholly caused by a lesion traumatic in origin (injury to the brachial plexus with traumatic arthritis of the shoulder) or whether syphilis of the nervous system was a factor in the causation of the condition.

Skiagrams.

DRS. J. G. AND W. A. EDWARDS displayed a series of interesting skiagrams depicting various pathological conditions. One series showed a normal lung followed by various stages of pneumoconiosis; another series depicted some unusual cases of obstruction of the large and small intestines.

Graham's method of outlining the gall bladder after the administration of sodium tetraiodophenolphthalein was well illustrated by several skiagrams.

Various diseased conditions of bones including *osteitis deformans*, congenital abnormalities and pathological changes in the head sinuses were also demonstrated.

THE AUSTRALASIAN MEDICAL PUBLISHING COMPANY, LIMITED.

ANNUAL MEETING.

THE ANNUAL GENERAL MEETING OF THE AUSTRALASIAN MEDICAL PUBLISHING COMPANY, LIMITED, was held at The Printing House, Seamer Street, Glebe, New South Wales, on August 24, 1926, DR. W. N. ROBERTSON, C.B.E., the Chairman, in the chair.

The following report of the Directors was presented and adopted.

The Directors submit their report for the past year and balance sheet as at June 30, 1926, together with profit and loss account for the twelve months ended June 30, 1926.

At the time of the last report the Company had recently (in the course of its development as owner and publisher of THE MEDICAL JOURNAL OF AUSTRALIA) entered upon a new stage of its career, namely, that of general printers. As was anticipated, seeing that this business was a new venture, the contracts and other work necessary to keep the printing machines running to their fully capacity took some time to accumulate. The machines have now been for some months working full time. They are still able to meet the demands made upon them, but extension of the plant is, nevertheless, being seriously contemplated.

The question of adding to the plant has arisen not only in anticipation of increased business coming in the ordinary way, but is due to the fact that it is proposed to produce an illustrated monthly popular health magazine designed to meet the requirements of outside readers for authoritative information on public health and medical subjects.

The proposal that the Company should undertake the publication of a popular health magazine came to it from the Federal Committee and it is noted that the report

(1925) of the Royal Commission on Health gave prominence to the suggestion as a means of educating the public in the principles of public health. In doing so, the Royal Commission recommended that the columns of the magazine should be made available for publishing information supplied by the Commonwealth Department of Health and the several State Departements.

In regard to THE MEDICAL JOURNAL OF AUSTRALIA it completed its twelfth year on June 30, 1926. In addition to recording and stimulating the growth of the medical and allied sciences in Australia, the journal has done much to satisfy the needs of the profession throughout the Commonwealth by keeping its members in close touch with the world of medicine and generally by fulfilling the functions of a medical newspaper.

Dr. W. N. Robertson, C.B.E., and Dr. H. S. Newland, C.B.E., D.S.O., retire from the Board of Directors in accordance with the Articles of Association and are eligible and offer themselves for reelection.

For the financial position of the Company the members are referred to the balance sheet and profit and loss account.

W. N. ROBERTSON,
August 24, 1926. Chairman.

Dr. W. N. Robertson and Professor F. P. Sandes were reelected Directors of the Company.

Obituary.

CHARLES VANCE SMITH.

WE regret to announce the death of Dr. Charles Vance Smith which occurred recently at Vienna, Austria.

SIDNEY VERE APPLEYARD.

WE have to announce with regret the death of Dr. Sidney Vere Appleyard which occurred at Lindfield, New South Wales, on August 28, 1926.

FREDERICK JOHN THOMAS SAWKINS.

WE regret to announce the death of Dr. Frederick John Thomas Sawkins which occurred at Goulburn, New South Wales, on August 30, 1926.

JOHN THEODORE ANDERSON.

IT is with much regret that we announce the death of Dr. John Theodore Anderson, late of Perth, Western Australia, which occurred at Melbourne on August 29, 1926.

Medical Appointments.

Dr. John Beasley Gillen (B.M.A.) has been appointed a member of the Gawler High School Council, South Australia.

* * * *

Dr. Harold Keith Pavy (B.M.A.) and Dr. Douglas Robson Wallman (B.M.A.) have been appointed members of the Naracoorte High School Council, South Australia.

* * * *

Dr. Herbert Leopold Ashton-Shorter (B.M.A.) has been appointed Government Medical Officer at Emerald, Queensland.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xxvi.

ADELAIDE HOSPITAL: Medical Registrar.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association.
QUEENSLAND: Honorary Secretary B.M.A. Building, Adelaide Street, Brisbane.	Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Honorary Secretary, 12, North Terrace, Adelaide.	Contract Practice Appointments at Ceduna, Wudinna (Central Eyre's Peninsula), Murat Bay and other West Coast of South Australia Districts.
WESTERN AUSTRALIAN: Honorary Secretary, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Diary for the Month.

SEPT. 14.—Tasmanian Branch, B.M.A.: Branch.
SEPT. 14.—New South Wales Branch, B.M.A.: Ethics Committee.
SEPT. 14.—Section of Medicine, New South Wales Branch, B.M.A.
SEPT. 15.—Western Australian Branch, B.M.A.: Branch.
SEPT. 15.—Central Northern Medical Association, New South Wales.
SEPT. 15.—South Sydney Medical Association, New South Wales.
SEPT. 15.—Section of Obstetrics and Gynæcology, New South Wales Branch, B.M.A.
SEPT. 20.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT. 21.—Tasmanian Branch, B.M.A.: Council.
SEPT. 21.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 22.—Victorian Branch, B.M.A.: Council.
SEPT. 22.—Western Medical Association, New South Wales (Annual).
SEPT. 24.—Queensland Branch, B.M.A.: Council.
SEPT. 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 28.—Illawarra Suburbs Medical Association, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.